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TUMORS OF THE NASOPHARYNX.

A Review of Fifty-Two Cases.*†

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I. INTRODUCTION.

Tumors of the nasopharynx, whether benign or malignant, constitute a perplexing problem for the otolaryngologist. The anatomical peculiarities of this area make adequate visualization and early diagnosis difficult; and by the time signs or symptoms caused by tumors of the nasopharynx occur, the tumor may have reached such an advanced state that the results of any type of therapy will be unsatisfactory.

Many papers on this subject have been written since the turn of the century. One of the earliest essays in this country was by C. Jackson[1] in 1901, in which he stated that he was able to find only 13 cases of nasopharyngeal carcinoma in the world literature. Godtfredsen,[2] in 1947, reported on a series of 454 cases of nasopharyngeal malignancy. As of this year, there have been perhaps a little over 3,000 cases of

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benign and malignant nasopharyngeal growths reported in the literature.

In this article, we plan to present our experiences with tumors of this area and compare them with those of other authors. We will also comment on some of the newer surgical and radiological approaches to the nasopharynx. It is our hope that this paper will serve to encourage our fellow otolaryngologists to search vigorously for the etiology of the vague complaints which so often are early signs of nasopharyngeal tumors. One of our biggest problems in the treatment of tumors of this area is, as previously intimated, the long delay from the onset of symptomatology until the pathological diagnosis can be made. Only by maintaining a high index of suspicion and by perseverance in the use of all of the diagnostic tools and methods available, can we overcome this problem.

In this report, 42 cases of primary malignant and 10 cases of primary benign growths of the nasopharynx will be reviewed. These cases were seen and diagnosed by the members of the staffs of the Departments of Otolaryngology and Radiology, at the University of Virginia Hospital, during the period 1935 through 1960. The records of over 150 patients with tumors of the nasopharynx were examined during the preparation of this paper. Over 100 cases were eliminated because it could not be definitely established that they were primary tumors of the nasopharynx, or else their records were not sufficiently complete to warrant their being reported. This review is a continuation of a study of all tumors of the upper respiratory tract seen and/or treated by members of the Departments of Otolaryngology and Radiology, at the University of Virginia Hospital, during this 25-year period.[2a]

II. INCIDENCE.

Tumors of the nasopharynx are relatively rare when compared with the incidence of tumors of the other areas of the body; however, when the frequency of tumors of the nasopharynx is compared to that of all tumors of the head and

neck regions only, this figure becomes more impressive. Our own figures (see Table I), taken from a five-year period (1955-1960), indicate that 0.6 per cent of all cancers diagnosed as such at the University of Virginia Hospital, were located in the nasopharynx. This figure compares favorably with those of Godtfredsen[2] (0.4 per cent) and Simmons and Ariel[3] (0.7 per cent) but is not so high as that of Pang[4]

TABLE I.

Incidence Studies: Cancers of Nasopharynx.

Race:		Sex:	
White	36 cases (85.7%)	Male 22 cases	Female 14 cases
Colored	6 cases (14.3%)	Male 2 cases	Female 4 cases
Ratio: 6.0 to 1 white to colored 4.0 to 3 male to female			
Age Incidence:			
1-19 yrs.	5 cases	50-59 yrs.	15 cases
20-29 yrs.	2 cases	60-69 yrs.	5 cases
30-39 yrs.	4 cases	70-79 yrs.	5 cases
40-49 yrs.	6 cases		
Average Age at onset			47.9 yrs.
Extremes of Ages			3-75 yrs.
Per Cent below 30 yrs. of age			16.7%
Per cent between 40 and 59 yrs. of age			50.0%

(1-3 per cent). Ackerman and Regato[5] report an incidence of 0.5-1.0 per cent of all cases of cancer as being primary in the nasopharynx. Martin and Blady[6] stated that cancer of the nasopharynx makes up about 2 per cent of all malignant growths seen in their Head and Neck Clinic. Ormerod[7] (London) estimated that 8 per cent of all malignancies seen in an Ear, Nose and Throat Clinic are found in the nasopharynx. Pang[4] reports that 3.5-5.8 per cent of all head and neck malignancies seen in two hospitals in Hawaii, were located in the nasopharynx.

Benign tumors of the nasopharynx are definitely less common than the malignant growths. In our own series (see Table II), there was a 4.2 to 1 ratio of malignant to benign growths in the nasopharynx. During the 25-year period covered in this paper, there were 331,957 admissions to our general hospital, which is part of a teaching institution. From

this figure, it may be estimated that one out of every 6,384 admissions had a tumor of the nasopharynx, while one out of every 7,904 admissions had a malignancy in this area. One out of every 33,196 admissions had a benign tumor of the nasopharynx, while one out of every 55,326 admissions had a juvenile angiofibroma (very similar to Handousa's[23] figure).

The average age of the patients with malignant tumors, in our series, at the onset of their symptoms was 47.9 years, with the extremes of ages being three years and 75 years.

TABLE II.

Over-all Incidence of Nasopharyngeal Cancers.

Incidence at the University of Virginia Hospital: 0.6% of all Cancers.
1 out of every 6384 admissions during 1935-60 had a tumor of the Nasopharynx.
1 out of every 7904 admissions during 1935-60 had a malignant tumor of the Nasopharynx.
1 out of every 33,196 admissions during 1935-60 had a benign tumor of the Nasopharynx.
1 out of every 55,326 admissions during 1935-60 had a juvenile angiofibroma of the Nasopharynx.

One-sixth (16.7 per cent) of our malignant cases were less than 30 years old at the time of onset of their symptoms, while 29 per cent were below the age of 40. These statistics agree with other series[1,2,3,4,6,8,9,10,11,12,13,14] for the most part.

Of the malignant tumors, 24 were in males and 18 in females, for a 4 to 3 ratio. In contrast, Molony[18] reports a 7 to 1 male to female ratio, while Hanckel states that there is a 2 to 1 male to female ratio. Another paper[9] reports an approximate 4 to 1 ratio.

Thirty-six of the patients were white, and six were colored (Negro), for a ratio of 6 to 1. (The ratio of white to non-white in the population served by our hospital is 4.5 to 1.) One-half (50 per cent) of the patients were in the age group from 40 to 59 years of age. There were no Chinese patients in our series.

The average age of patients with benign tumors, excepting those with juvenile angiofibromata, was 40 years, with extremes of 7 to 69 years of age. The average age of patients with juvenile angiofibromata at the time of onset of symptoms was 14.2 years, with extremes of 10 to 19 years of age. All of the cases with juvenile angiofibromata were white males. There was a 9 to 1 ratio of white to non-white patients with benign tumors. Schiff[21] states that the estimated incidence of juvenile nasopharyngeal angiofibroma is .05 per cent of all neoplasms of the head and neck.

III. HISTOPATHOLOGY.

Histopathologic classification of malignant tumors of the nasopharynx has been a rather confusing problem, and this in turn has tended to make comparative morbidity statistics

TABLE III.

Histopathological Classification: Cancer of Nasopharynx.

Classification:	
Transitional Cell Carcinoma	19 cases (45%)
Epidermoid Carcinoma	13 cases (31%)
Lymphosarcoma	3 cases (7%)
Other	7 cases (17%)
Total	42 cases

somewhat unreliable. Godtfredsen,[15] Scanlon, Devine, and Woolner,[9] Korkis,[16] and Eggston,[17] plus others, have discussed the problems associated with the classification of tumors of this area. In our own series of malignant nasopharyngeal tumors, we have classified them as follows (see Table III): Transitional cell carcinoma which includes lymphoepitheliomas, 19 cases; epidermoid (squamous cell) carcinoma, 13 cases; lymphosarcoma, 3 cases; and others which include 3 cases of anaplastic carcinoma, 1 case of adenocarcinoma of salivary gland type, 1 case of embryonic rhabdomyosarcoma, 1 case of adenocystic carcinoma, and 1 case of hemangioendothelioma. Eighty-eight plus per cent (88.1 per cent) of our malignant tumors were thus carcinomas, which is a slightly lower figure than Pang's,[4] 94 per cent. Other tumors which

have been reported as being in the nasopharynx include lymphomas, cylindromas, extramedullary plasmocytomas, and chordomas. (Most chordomas are benign, but about 11 per cent are known to have metastasized.)[19]

Benign tumors of the nasopharynx may include juvenile angiofibroma, mucous cysts, lipoma, neurofibroma, mixed tumors of the salivary gland type, chondroma, xanthoma, sarcoid, ossifying fibroma, myxofibroma, and teratoma. Several of these benign tumors may be considered locally malig-

TABLE IV.

Histopathological Classification: Benign Tumors of Nasopharynx.

Juvenile Angiofibroma	6 cases
Myxofibroma	1 case
Ossifying Fibroma	1 case
Sarcoid	1 case
Polypoid Fibrous Chronic Granulation Tissue	1 case
Total	10 cases

Average Age at Time of Diagnosis:

Juvenile Angiofibroma	14.2 years (Extremes 10-19 yrs.)
All other benign tumors	40.0 years (Extremes 7-69 yrs.)

Only one case of benign nasopharyngeal tumor was a non-white patient.

nant, *e.g.*, juvenile angiofibroma and myxofibroma. In our own series (see Table IV) of 10 benign tumors, six were juvenile nasopharyngeal angiofibromas, and there was one each of the following: sarcoid, ossifying fibroma, myxofibroma, and one reported as polypoid fibrous chronic granulation tissue.

IV. RELATED ANATOMY.

The nasopharynx (epipharynx, nasal part of the pharynx, or nasal pharynx) is a six-walled irregular cavity located behind the choanae, above the pharyngeal isthmus at the level of the palate, below the base of the skull, anterior to the first two cervical vertebrae and basilar portion of the occipital bone, and medial to the Eustachian tube orifices.

The lateral walls of the nasopharynx contain the Eustachian

or auditory tube orifices, above and behind which are the fossae of Rosenmuller or so-called pharyngeal recesses. One author[15] has reported that as high as 50 per cent of the nasopharyngeal malignancies originate in the lateral wall of the nasopharynx, especially in the fossa of Rosenmuller. Most observers agree that the fossa of Rosenmuller is the commonest primary site for malignancies of the nasopharynx.

The floor of this cavity is formed by the superior surface of the soft palate. Simmons and Ariel[3] state that only seldom does cancer originate from the floor or anterior wall of the nasopharynx, and our own experience agrees with this.

The pharyngeal aponeurosis, or pharyngeal fascia, which is entirely closed and rather resistant to the spread of tumor or infection, is continuous with the fibrocartilaginous tissue filling the foramen lacerum. It is through the foramen lacerum and the nearby foramen ovale that tumors find the route of least resistance into the middle cranial fossa. Tumors spreading by these routes immediately come into contact with the cavernous sinus and/or the gasserian ganglion and its branches. Lying closer to the fossa of Rosenmuller than the Vth or VIth nerves is the vidian nerve or nerve of the pterygoid canal. The abducens nerve (VIth) passes through the cavernous sinus lateral to the internal carotid artery. This cranial nerve is the one most commonly involved in intracranial spread of nasopharyngeal malignancies. Cranial nerves II, III, IV, and V also lie in close proximity to VI and are sometimes involved.

Other anatomical relations to the nasopharynx are the close association of the occipital bone, sphenoid sinus, and the infratemporal fossa (or maxillopharyngeal space) which is located anterior to the Eustachian tube orifice and extends laterally to the vertical ramus of the mandible. Included in the maxillopharyngeal space are the mandibular and maxillary nerves. Another area, located behind and lateral to the nasopharynx, in close proximity to the fossa of Rosenmuller, is called the retroparotid space by Ackerman and Regato.[5] Included in this space are cranial nerves IX, X, XI, and XII, the internal carotid artery, internal jugular vein, and the cervical sym-

pathetic chain; thus, tumor invasion in this space may cause a Horner's syndrome, hemiparalysis of the soft palate and of the wall of the pharynx, and hemiparalysis of the tongue and larynx.

The lymphatic drainage from the nasopharyngeal area is to the retropharyngeal nodes, the upper internal jugular nodes, the spinal accessory nodes, and the supraclavicular nodes.

TABLE V.

Signs and Symptoms: Cancer of Nasopharynx.

List of Signs and Symptoms in Order of Frequency:	
Enlarged Cervical Nodes	25 cases (60%)
Blockage of Ears (Hypoacousia)	22 cases (52%)
Bloody Nasal Discharge	21 cases (50%)
Weight Loss	20 cases (48%)
Head and Neck Pains	17 cases (40%)
Nasal Obstruction	16 cases (38%)
Secretory Otitis Media (middle ear effusion)	10 cases (24%)
Sore Throat	8 cases (19%)
Diplopia	5 cases (12%)
Voice Change	5 cases (12%)
Tinnitus	4 cases (10%)
Trismus	4 cases (10%)
Cranial Nerve Palsy (Other than VI)	3 cases (7%)

14 cases (35%) had bilateral cervical metastases.

Frequency of Signs and Symptoms as presenting evidence of nasopharyngeal cancer.

Enlarged Cervical Nodes	14 cases (35%)
Head and Neck Pains	7 cases (17%)
Hypoacousia	7 cases (17%)
Nasal Obstruction	6 cases (14%)
Bloody Nasal Discharge	3 cases (7%)
Secretory Otitis Media	3 cases (7%)
Sore Throat	2 cases (5%)

V. SIGNS AND SYMPTOMS.

Malignant tumors of the nasopharynx produce a variety of signs and symptoms (see Table V). Those most often seen in the order of their frequency were: *1.* enlarged cervical nodes, *2.* blockage of ears or hypoacousia, *3.* bloody nasal discharge, *4.* weight loss, *5.* head and neck pains, *6.* nasal obstruction, *7.* secretory otitis media, *8.* sore throat, *9.* diplopia, and *10.* voice change. When we recorded the frequency of signs and symptoms which were the presenting or first

evidence of nasopharyngeal malignancies, we found that the commonest was an enlarged cervical node (or nodes) followed by head and neck pains, hypoacousia, nasal obstruction, bloody nasal discharge, secretory otitis media, and sore throat, in that order. It should be pointed out that the difference in these two groupings is that the first group in Table IV represents all of the signs and symptoms seen in the nasopharyngeal malignancies, while the second grouping consists only of that sign or symptom which the patient or his physician *first* noticed in the course of the disease.

The importance of either a unilateral or bilateral painless enlarged cervical node as a sign of nasopharyngeal malignancy cannot be stressed too often. Sixty per cent of our patients had enlarged metastatic nodes, and in 35 per cent of our cases, the nodes were bilateral. The commonest sites for cervical metastasis were the upper internal jugular chain, the subdigastric nodes, and the spinal accessory nodes or chain.

The feeling of fullness in the ears with slight to marked decrease in auditory acuity was present in 52 per cent of our cases at some stage of their disease, but was the presenting symptom in only 17 per cent of the cases. Secretory otitis media, on the other hand, was present in 24 per cent of our patients during the course of their disease, and was a presenting symptom in 7 per cent. Both of these conditions responded coincidentally to X-ray or cobalt therapy with marked improvement in most instances. Because of the relative frequency with which hypoacousia and secretory otitis media appear in nasopharyngeal malignancies, the otolaryngologist should ever be alert to carry out a careful, complete, nasopharyngeal examination when his patient complains of these symptoms.

Bloody nasal discharge and nasal obstruction occurred in 50 per cent and 38 per cent of our cases, respectively. They were presenting symptoms in 7 per cent and 14 per cent. Weight loss was a surprisingly common sign, being present in 48 per cent of our patients. The average weight loss in the 20 individuals complaining of this sign was 22.2 pounds. As far as we could ascertain from the records, this weight

loss was due to local factors (pain, sore throat, dysphagia) rather than to any systemic effect of the cancer in most instances.

There were only five cases (12 per cent) with diplopia or VIth nerve involvement. Forty per cent of our patients had various head and neck pains, an undetermined number of which had Vth nerve complications. Cranial nerves II, III, IV, V, VI, VII, VIII, IX, X, XI, and XII were involved in one or more of our cases. Ten per cent of those surveyed had trismus, due to either direct extension into the pharyngo-maxillary space or a metastatic node in this region. Korkis'

TABLE VI.

Signs and Symptoms: Benign Tumors of Nasopharynx.

Nasal Obstruction	6
Nose Bleed	4
Anterior Nasal Drainage	2
Weight Loss	2
Voice Changes	2

Duration of Symptoms prior to diagnosis:

Juvenile angiofibroma: 6.6 months (average)
Others: Varied from 7 months to 10 yrs.

Text[16] states that the most likely order of neural involvement would be the vidian nerve, maxillary nerve, and abducens nerve. Flatman's[20] detailed anatomical studies of the relationship of the cranial nerves to the roof of the nasopharynx and fossa of Rosenmuller showed the vidian nerve or nerve of the pterygoid canal to be closer to the fossa of Rosenmuller than the Vth or VIth nerves. One of the symptoms of dysfunction of the vidian nerve is lacrimation. Both Korkis[16] and Flatman[20] suggested that lacrimation might be an early sign of a nasopharyngeal malignancy, but that it had not been accurately recorded as such in the medical histories of cases of nasopharyngeal malignancies. There were only two patients in our series who had pronounced lacrimation, but a more careful history in each case might have revealed this symptom to be more common.

Benign tumors of the nasopharynx produce a more constant

group of signs and symptoms (see Table VI) than the malignant tumors. This is due to the fact that benign tumors show their presence by localized growth and pressure, and in the case of the angiofibromata, by excessive vascularity. The patients in our series presented the following manifestations in order of frequency: nasal obstruction, epistaxis, anterior nasal drainage, weight loss, and voice changes. Schiff,[21] in his paper on juvenile nasopharyngeal angiofibroma, says that the signs and symptoms of the angiofibroma usually have a march of progression and start with nasal obstruction, followed by recurrent epistaxis, progressive deformity of the palate, face and throat, rhinolalia, and anosmia. His obserservations are very similar to our own findings, as well as others,[22,24,25,26,27,28,29], in this type of benign tumor. Other benign tumors of the nasopharynx, in our series, caused various symptoms, including otalgia, snoring, hearing loss, and sore throat.

VI. DIAGNOSIS.

A high index of suspicion and the astute use of available tools and methods are necessary to accomplish an early diagnosis of a nasopharyngeal malignancy. Once the full picture has developed, localization of the tumor is no problem; but adequate therapy is then exceedingly difficult. Our main plan of attack should be aimed at determining the diagnosis early, as this appears to be the phase in which we have lagged the most. For example, in our series, there was an average interval of 9.4 months from onset of symptoms to establishment of diagnosis (see Table VII). Others[3,4,10,30,31] report from 6.1 to 12 months as the average duration of symptoms prior to diagnosis. In an attempt to analyze the reasons for this delay in diagnosis, we found that the patient, in many cases, looked upon his or her symptoms as nothing serious and thus did not seek medical advice promptly. On the other hand, the physician, even when consulted early, sometimes failed to attach the proper significance to rather ominous signs in their patients, *e.g.*, treatment of painless cervical adenopathy with antibiotics, tonsillectomy for head and neck pains, biopsy of cervical node without examination of nasopharyngeal, oral,

and laryngopharyngeal spaces, etc. As noted before, the presenting or first sign of nasopharyngeal malignancy in 33 per cent of our cases was a metastatic cervical node. In this situation, early diagnosis is thus impossible, but there should be no further delay. We agree with Ackerman and Regato[5] that patients with metastatic tumor of the upper cervical region should be assumed to have a primary nasopharyngeal lesion unless otherwise demonstrated.

Rarely, a completely asymptomatic lesion may be discovered during a routine, careful, nasopharyngeal examination. The

TABLE VII.

Incidental Statistics: Cancer of Nasopharynx.

Interval from onset of symptoms to diagnosis	Average	9.4 months
Interval from onset of symptoms to death	Average	26.7 months
Interval from onset of treatment to death	Average	15.1 months

57.1% of tumors were located on the right side of the nasopharynx initially.
38.1% of tumors were located on the left side of the nasopharynx initially.
4.8% of the tumors were in the midline or bilaterally initially.

Average Weight Loss in 20 Patients was 22.2 pounds.

Average Hemoglobin determination in 17 patients was 12.8 gms %.
Average Hematocrit determination in 18 other patients was 41.5%.

Therefore anemia was not a common finding in Cancer of the Nasopharynx.

methods of examining the nasopharynx include the use of the nasopharyngeal mirror (probably the best and most reliable), the nasopharyngeal or Yankauer speculum, the electric nasopharyngoscope, digital palpation, and anterior and posterior rhinoscopy. In addition, X-rays, which include lateral views of the nasopharynx, a base view of the skull, and also stereoscopic films, often help in establishing the diagnosis and extent of the tumor. The conclusive diagnosis, however, can be made only by histopathologic examination of a tissue biopsy specimen. If no tumor is seen on examination and a nasopharyngeal tumor is suspected, a biopsy, especially in the fossa of Rosenmuller, is of particular value. In the past several years, we were asked to examine a number of patients with meta-

static cervical nodes with unknown primary. In three cases with what appeared to be a completely normal nasopharynx, a positive diagnosis of nasopharyngeal carcinoma was obtained by random biopsy. Cytologic examination has been suggested[22] as an aid to early diagnosis of lesions in this region, but as yet we have not used this method in this particular area.

In establishing the tissue diagnosis of a suspected juvenile angiofibroma, one should proceed with caution and be prepared to stop the rather brisk hemorrhage which usually follows a biopsy. This means that posterior nasal packs or nasopharyngeal balloons with sufficient material for anterior nasal packing should be readily available. In some cases, it may be wise to perform the biopsy in the operating room under general anesthesia.

TABLE VIII.

Maxims of Therapy for Cancers of the Nasopharynx.

I. Irradiation remains the treatment of choice for Primary Cancers of the Nasopharynx. This Irradiation may be given by any of the external forms now available which include conventional X-ray, supervoltage X-ray, and cobalt 60 sources.
II. External irradiation may be augmented by the following: (a) Intercavitary and Interstitial Irradiation. (b) Transpalatal surgery and fenestration. (c) Radical Neck Dissection (uni- and bilateral). (d) The newer perfusion techniques and adjunctive chemotherapy.

VII. THERAPY.

The primary treatment of malignant tumors of the nasopharynx is external irradiation (see Table VIII), and this maxim of therapy is universally accepted.[2,3,4,5,6,8,9,10,11,12,13,14,16,17,18,19,33,34] Surgery, as the primary treatment for cancer in this area, is highly unsatisfactory for many reasons, the most impressive being the fact that complete surgical extirpation of malignant lesions of the nasopharynx is virtually impossible. (There have been no reports of lasting cures by this method.)

The external irradiation may be administered by conven-

tional X-ray therapy, super-voltage X-ray, or cobalt 60 teletherapy (see Table IX). In general, external irradiation is given to the nasopharynx and upper cervical region of all patients with primary malignancies of the nasopharynx, even when there is no objective evidence of cervical metastases. When there are cervical metastases, a wide field is irradiated from the base of the skull to the clavicle. Kramer[11] states that radiotherapy must aim at eradicating the disease with the first course of treatment, as recurrent cases can hardly

TABLE IX.

Summary of Therapy: Cancer of Nasopharynx.

Malignant Tumors (Cancer)
Three cases had surgery initially.
Two cases had surgery secondarily.
Extremes of Primary Therapy using External Irradiation: 1650-9000r.
Average Dose of Primary Therapy using External Irradiation: 4500r.
20 Patients had to receive secondary courses of irradiation.
Forms of Irradiation Therapy Used in This Series:
Cobalt 60 Teletherapy.
250 KV Conventional X-ray.
Intercavitary Radium.
Interstitial Radon Seed Implants.

ever be cured. For this reason, the tumor dosage should not be less than 5500-6000 r, initially. Even higher dosages may be required to effect a cure, but the patient may have to pay the penalty for the cure with cord damage, radiculitis, skin damage, bone necrosis, etc. We have given up to 9000 r of external irradiation to one patient, and a total of 16,000 r of external and intercavitary-interstitial irradiation in another case, without excessive reaction. Another patient received 19,066 r of external irradiation to the nasopharynx and cervical nodes, plus an additional 3500 r of intercavitary radiation over a period of three and one-half years without untoward side effect.

Surgery for malignant tumors of the nasopharynx is limited primarily to that necessary to establish diagnosis, to provide access and visualization (palatal fenestration), occa-

sional radical neck dissection, and, rarely, excision or electrodesiccation of the primary lesion.

The palatal fenestration procedure, as described and advocated by Wilson,[35,36] is one surgical procedure which has very definite merits in the management of nasopharyngeal cancers. By this operation, a permanent "window" is constructed in the palate, which allows direct visualization of the nasopharynx. Through this fenestra, the location, size, and extent of the tumor can be determined at the time of the operation and thereafter. This aids the otolaryngologist and the radiotherapist in planning the direction, dosage, and effect of the external irradiation throughout the course of therapy and in subsequent follow-up examinations. Recurrences may be discovered earlier, and adequate biopsy of suspicious areas may be performed more easily through the palatal fenestra. The wearing of a dental prosthesis (which should be made prior to the operation) to cover the defect is the only disadvantage to the patient, and this is certainly a small price to pay for the advantage obtained.

Radical neck dissection for cancer of the nasopharynx has been advocated in certain selected cases,[4] but most authors[5,16,39,40] feel that this procedure is not indicated, because it is not possible to obtain an *en bloc* resection with the primary and intervening tissue. It is our feeling that radical neck dissection may be of value to the patient whose primary has been controlled by irradiation (as evidenced by negative examination and biopsy, lack of cranial nerve involvement, no cranial bony changes by X-ray) but whose cervical node metastases have failed to respond to irradiation. Prophylactic neck dissection probably is of little benefit because of the inability to obtain an *en bloc* resection, the frequency of intracranial metastases, the relatively high frequency of bilateral cervical metastases, and the relatively high radiosensitivity of these metastatic nodes. Pang[4] reports on two cases of neck dissection, performed by him, which had had previous deep X-ray therapy to the cervical region. In these cases, a careful microscopic examination of the neck contents did not reveal any residual carcinoma.

Another newer form of therapy, which has shown encouraging results in certain head and neck malignancies, is the perfusion technique, popularized by Creech[37] and his associates and, more recently, by Sullivan[38] *et al.* In the perfusion technique for head and neck cancers, an anticancer drug, such as nitrogen mustard, phenylalanine mustard, actinomycin D, or others, is injected through the isolated external carotid artery (or arteries) in exceptionally high dosages and then recovered on the venous side through the internal jugular vein, thus avoiding the systemic effect of the high dosages of the toxic drugs used. The pump oxygenator has to be utilized with this technique. As can be seen, regions thus isolated can be perfused with very high concentrations of anticancer agents, with only local toxicity resulting in most instances.

In Sullivan's technique, the pump oxygenator is not required. He introduces an antimetabolite continuously through the external carotid artery (or arteries) but makes no attempt to recover the agent, as mentioned in the other technique. Instead, he injects a specific antidote intramuscularly at intervals, and this prevents the severe systemic effects of the antimetabolite. In his presentation to the American Society for Head and Neck Surgery, in October 1960, Sullivan reported some rather favorable results from the use of the antimetabolite-metabolite technique in epidermoid carcinoma of the head and neck regions. It goes without saying that these techniques are not without danger to the patient, as a few have died from complications of the treatment. Certainly, chemotherapy for cancer of the nasopharynx offers much promise for the future, and we should expect to see newer and more effective drugs and techniques of administration resulting from the intensified research in this field.

Whereas irradiation is the treatment of choice for malignant tumors of the nasopharynx, surgery is the treatment of choice for most all of the benign tumors of this area. The various adjuncts to surgical removal of benign neoplasms include irradiation, sex hormones, electrocoagulation, and local chemical therapy.

The juvenile angiofibroma of the nasopharynx is probably

the commonest benign neoplasm in this region, and our discussion of therapy will deal mainly with this tumor. Surgical excision of nasopharyngeal angiofibromas is the treatment of choice, once the diagnosis has been established. Although most of these tumors will usually involute by the time the patient reaches 25 years of age, this is a dangerous and potentially disfiguring tumor if not treated promptly and adequately. The danger of severe hemorrhage, nasal obstruction, rapid local extension into the surrounding structures, and the marked permanent disfigurement which may result from delaying therapy are certainly strong arguments for prompt treatment. Of course, each case must be evaluated individually, and the time and type of therapy may have to be varied according to the situation. With the advent of hypothermia-hypotensive anesthesia, the excision of these extremely vascular tumors is less of a problem than formerly.

Schiff[21] has presented evidence that the administration of stilbestrol, 5 mgm. t.i.d. for one month, will cause involution of the angiofibroma as well as a decrease in the vascularity. He suggests that the use of estrogen therapy is worth trying as a supplement to other methods, especially prior to surgery. We have tried this form of pre-operative preparation in one case, but our pathologists were unable to demonstrate any decrease in the vascularity by comparison of the microscopic slides of the pre- and post-stilbestrol tissue specimens. We used hypotensive-hypothermia anesthesia in this same case, and thus the decrease in blood loss which occurred could not be ascribed entirely to utilizing the hormone therapy or to the type of anesthesia.

Androgen therapy has been recommended,[41] but this has produced few dramatic involutions of the tumor. Some authors[29] believe that androgens used in combination with irradiation may enable the administration of smaller doses of irradiation to control the growth of the angiofibroma. For the most part, androgenic therapy for the control of juvenile angiofibromas has been disappointing.

External, interstitial, and intercavitary irradiation has been utilized[24] for definitive treatment and also preoperatively to

decrease the vascularity by causing an obliterative endarteritis. The dangers of irradiation therapy, especially in patients who have not reached full facial bone growth, should be remembered. Extensive irradiation is to be avoided. There is a case report[42] of a fibrosarcoma arising in a juvenile nasopharyngeal angiofibroma following extensive irradiation. We do not advocate irradiation therapy as the primary form of treatment for angiofibromas of the nasopharynx.

Electrocoagulation is recommended for use in the case of a small recurrence, but is not so effective as surgical excision in the primary treatment of this tumor.

Many surgical approaches to the nasopharynx have been described and used. These include such avenues as the intra-

TABLE X.

Histopathological Classification and Correlated Survival Statistics: Cancer of Nasopharynx.

	2 years	5 years	10 years
Transitional Cell Carcinoma	7	5	1
Epidermoid Carcinoma	3	2	0
Lymphosarcoma	2	2	2
Other	2	1	0
Total:	14 (35.9%)	10 (27.0%)	3 (10.0%)
Determinate Cases:	39	37	30

oral, the transantral via a Caldwell-Luc or Denker procedure, the intranasal, the transpalatal, the wide field via the Weber-Ferguson incision, and the mandible-splitting lateral neck incision. When the tumor is limited to the confines of the nasopharynx, we prefer the combined transantral and intraoral approach. The wider or more radical exposures are required when the angiofibroma has extended into the adjacent areas, *e.g.*, the pterygomaxillary space.

VIII. RESULTS OF THERAPY.

Out of 37 determinate cases with nasopharyngeal malignancies, ten have survived five years or longer for a survival rate of 27.0 per cent (see Table X). This figure is similar

to those of Martin and Blady,[6] Kramer,[11] Godtfredsen,[2] and Lenz.[43] In 39 determinate cases, we have a two-year survival rate of 35.9 per cent; and of 30 determinate cases, we have a ten-year survival rate of 10 per cent (three cases).

All but one of our 42 cancer patients have received irradiation as the primary form of treatment. In three of these cases, surgery was performed initially, and two had surgery secondarily. (Of these five patients, only one survived as long as two years, and all were dead before three years.) From our results, it would appear that patients with lymphosarcoma of the nasopharynx have the best prognosis, followed in turn by those with transitional cell carcinoma or lymphoepitheliomas.

Findings which markedly affected the prognosis or survival rates adversely were: evidence of bony involvement, cranial nerve involvement, and distant metastases. Eberhard and Leaming[44] report that almost 40 per cent salvage can be expected if there is no evidence of bony involvement.

Treatment given to our patients with benign tumors of the nasopharynx has apparently been effective in all but two instances. One of these has a myxofibroma which has extended well out of the confines of the nasopharynx into the neck, in spite of repeated surgical excisions; also, one of our cases with a juvenile angiofibroma has now had seven operations, including one radical mandible-splitting procedure, and still is not completely controlled. There has been one patient with a juvenile angiofibroma of the nasopharynx, at this hospital, whose diagnosis was made and treatment carried out prior to the beginning of this survey (1935-1960), who had 24 operative procedures between the ages of 14 and 25 years and three procedures after age 25. He is alive and well at 58 with no evidence of any abnormality of the nasopharynx. We have had no fatalities from benign tumors of the nasopharynx or from the therapy given for them.

IX. SUMMARY.

A review of 42 cases of primary malignant and ten cases

of primary benign tumors of the nasopharynx has been presented.

Six-tenths per cent of all malignant tumors diagnosed at the University of Virginia Hospital were located in the nasopharynx. There was a 4 to 3 male to female ratio of patients with cancer of the nasopharynx in this series, and a 4.2 to 1 malignant to benign tumor ratio.

The commonest malignant tumor was the transitional cell carcinoma (which included lymphoepithelioma), and the next most frequent was the epidermoid carcinoma. The commonest benign tumor in this area was the juvenile angiofibroma.

The anatomical features of the nasopharynx, as related to symptomatology produced by tumors of this area, have been briefly presented. Lacrimation, as a very early sign of nasopharyngeal malignancy, is emphasized and explained.

The most usual signs and symptoms of malignant nasopharyngeal tumors, in the order of their frequency, were: *1.* enlarged cervical nodes, *2.* hypoacousia, *3.* bloody nasal discharge, *4.* weight loss, *5.* head and neck pains, and *6.* nasal obstruction. Benign tumors of this area produced the following symptoms and signs in order of frequency: *1.* nasal obstruction, *2.* nose bleed, *3.* anterior nasal drainage, *4.* weight loss, and *5.* voice changes.

An average delay of 9.4 months from onset of symptoms to establishment of diagnosis, in malignant tumors of the nasopharynx, is reported.

The treatment of choice for cancer of the nasopharynx is external irradiation, using either conventional X-ray, supervoltage X-ray, or cobalt 60 teletherapy. The newer perfusion techniques and intra-arterial antimetabolite infusion treatment for malignancies of the nasopharynx offer an additional and sometimes very successful form of therapy for our consideration. Surgery, including radical neck dissection, is rarely indicated except in the case of the palatal fenestration procedure.

Surgical excision is recommended for the juvenile angio-

fibroma of the nasopharynx. The use of estrogenic compounds preoperatively is discussed.

A five-year survival rate of 27.0 per cent is reported in the cases with cancer of the nasopharynx.

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SIXTH INTERNATIONAL CONGRESS OF AUDIOLOGY.

The Sixth International Congress of Audiology will be held in Leyden, The Netherlands, September 5-8, 1962.

President: Prof. Dr. H. A. E. van Dishoeck;

Secretary: Dr. A. Spoor.

The program will include three round-table talks on "Frequency analysis of the normal and pathological ear." Moderator: Prof. Dr. G. von Békésy. "Central deafness in children." Moderator: Prof. Dr. J. M. Tato. "Psychogenic deafness and simulation." Moderator: Prof. Dr. H. A. E. van Dishoeck, and associated and independent papers.

Official languages of the Congress are: English, French, German and Spanish. Working languages will be: English and French.

For further information address the secretariat, Ear-Nose-Throat Department, Academisch Ziekenhuis, Leyden (The Netherlands).

BILATERAL CHOANAL ATRESIA IN THE NEWBORN— A NEW METHOD OF MEDICAL MANAGEMENT.*†

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Danville, Virginia.

Through the efforts of Beinfield[1] and others, the role of bilateral choanal atresia in the problem of severe respiratory distress in the newborn has been clarified. This congenital anomaly, however, is still not universally recognized; the routine testing of the patency of the posterior nares being seldom done. A surprising number of physicians are unaware of the strong instinctive use of the nasal airway in the newborn. In the presence of complete atresia of the posterior choanae the infant will exert every effort to the point of asphyxia to establish a nasal respiratory cycle. The newborn appears unable to prevent suffocation by adopting an oral air pathway on his own initiative.

Mouth breathing is an acquired habit. The aversion to mouth breathing is based on the evolutionary need for air currents to pass over the olfactory area.[2] Only rarely will the newborn spontaneously use his oral airway, but most infants can be trained to mouth breathing in three to four or more weeks.

It is generally agreed that bilateral choanal atresia in the newborn is an emergency surgical situation. To avoid asphyxia and to establish a feeding pattern, it is urgently necessary to obtain a satisfactory nasal airway. Recently Fitz-Hugh and I[3] have discussed the merits and techniques of the transnasal, transeptal and transpalatine operation in the surgical management of choanal atresia.

Under certain circumstances immediate surgery is inad-

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visable. In the presence of an associated congenital anomaly, such as congenital heart disease,[4] when prematurity is a factor, or when exhaustion and inanition intervene as a result of a delayed recognition of the condition, medical management is preferable. Definitive surgery can then be performed at a time when the operative risk is improved. Tracheostomy is seldom necessary.

Any surgical procedure on the newborn is fraught with difficulties; the size factor alone is formidable. In the case

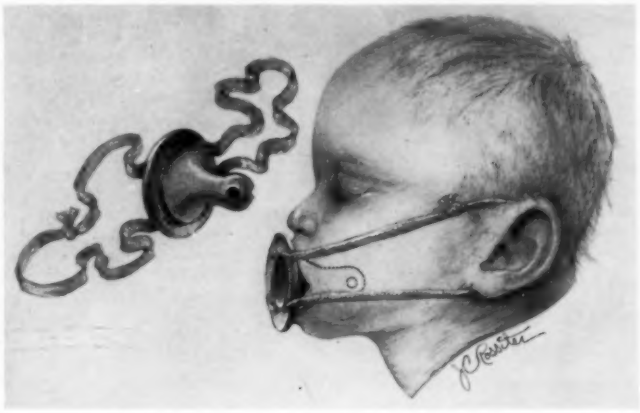

Fig. 1.

of choanal atresia, if the surgical correction can be deferred for a year, the operative area is twice the size of that of the newborn.

I have recently had the opportunity to observe the use of a simple device invented by the parents of a child born with bilateral choanal atresia. The success achieved in this case with an elementary nipple has prompted this report. With its use the problem of the child's respiratory distress was easily solved, and a mouth breathing habit was soon developed. In addition, the device proved to be the solution to the infant's

feeding difficulties; the mother periodically filled the bowl of the nipple with milk, the infant suckled and then was allowed to breathe through the enlarged holes in the nipple. The process was repeated until the required amount of the formula was consumed.

As illustrated, the device is a simple nipple. Tape is threaded through the rim of the nipple and fastened around the child's ears, holding it snugly into the mouth. For the purpose of breathing and eating, a single large hole or two lateral holes are made in the apex of the nipple. In the first weeks of life constant attention is necessary to avoid displacement of the nipple.

CASE REPORT.

According to the parents the child was born without difficulty after an uneventful pregnancy. Soon after birth the child presented the cardinal signs of bilateral choanal atresia—cyclic cyanosis, respiratory distress on feeding, and intermittent anoxia relieved by crying. It is not known when the precise diagnosis was made, but the urgent symptoms of respiratory difficulty necessitated a tracheostomy, followed later by an intranasal correction of the choanal atresia; the operation was probably handicapped by the very small size of the nasal fossae. Temporarily the nasal airway seemed adequate, and the tracheostomy opening was closed.

In the succeeding days difficulty was experienced in keeping the nasal airway patent; repeated attempts were made to maintain the openings made in the atresia. In spite of many dilations the posterior nares closed completely.

The patient was discharged from the hospital after giving the parents careful instructions on the child's care and was referred to me for further surgery. During the interval before attending my office the parent originated this method of keeping the baby's mouth open.

On his first visit to my office the child was breathing comfortably through the nipple, and the parents demonstrated the ease with which he was fed. The posterior choanae were entirely occluded. The baby appeared to be flourishing and gaining weight. He did not show any associated anomaly. Because of the patient's steady improvement the parents were reluctant to go through with another operative ordeal, and indeed there was no immediate need for surgery.

The baby now has developed complete mouth breathing. It is planned at a later date to reoperate, using the transpalatine route.

CONCLUSION.

A simple nipple device is described. The instrument is designed to assist in the medical management of bilateral choanal atresia in the newborn. The use of the nipple in a

single case was considered to be of sufficient success in solving the breathing and feeding problem to warrant this report.

The advantages of a method of medical management in a condition which can only be corrected surgically, is to allow time for the newborn infant to mature in size and strength and allow the surgeon optimal conditions for the successful correction of an uncommon congenital anomaly.

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SOUTHERN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY.

The section of Ophthalmology and Otolaryngology of the Southern Medical Association announces that it is now accepting papers by physicians of either specialty, living in the area of the Southern Medical Association, for consideration for presentation at the next annual meeting to be held in Dallas, Texas, from November 6 to 9, 1961.

The paper or an abstract of the paper may be sent directly to the Secretary, Dr. Albert C. Esposito, Suite 1212, First Huntington National Bank Building, Huntington, West Virginia, as soon as possible.

PERSONAL EXPERIENCES WITH THE SHEA OVAL WINDOW-VEIN GRAFT TECHNIQUE.

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Houston, Texas.

Stapedectomy is rapidly becoming the operation of choice for restoration of hearing in patients with otosclerosis. Hearing results have been exceedingly good, and complications have been few. There have been occasional postoperative cochlear losses, and these, of course, have caused some concern. Although total removal of the stapes was attempted by Hall and Rytzner in 1955,[2] the vein graft with polyethylene prosthesis operation, developed by Shea,[8] was the first successful stapedectomy procedure for restoration of hearing to near normal levels. Early reports of results of stapedectomy in combination with various prostheses have been important in the generation of enthusiastic support for the procedure.[4,5,6,7]

After careful study of our results with stapes mobilization and its modifications and the known limitations of fenestration of the semicircular canal, stapedectomy appeared to be the logical approach to the problem.[1] Observation of the method as performed by Shea, and verbal reports of his results, indicated that the operation was a safe, efficacious procedure when meticulously performed; consequently, between December, 1958 and November, 1960, 414 stapedectomies with vein grafts were performed. This report is based on this series of patients.

OVERALL HEARING RESULTS.

The superiority of the Shea vein graft operation as compared to stapes mobilization and fenestration of the labyrinth

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is illustrated (see Fig. 1). The improved hearing after stapedectomy with vein graft is evident with regard to closure of the bone-air gap, initial failure, refixation and closure of the fenestra; however, the complication of additional postoperative hearing loss which may occasionally occur after stapedectomy must also be considered.

METHOD OF REPORTING RESULTS.

It is important to note the inadequacy of reporting postoperative stapedectomy hearing losses as a complication of

TABLE I.

The Complication of Additional Hearing Loss Following Stapedectomy.

1. Based on Pure Tone Tests Alone	
Total Number of Patients	Number of Losses
382	3—0.7%
2. Based on Discrimination Tests	
Severe Losses (Additional Loss of More Than 20% with Final Score Less Than 50%)	
382	7—1.9%
Partial Losses (Additional Loss of Less Than 20% with Final Score Below 75%)	
382	4—1.0%

the procedure when the hearing is assessed only on the basis of pure tone audiometry. When pure-tone testing alone is considered, only 0.7 per cent of our patients showed increased hearing loss as a complication of the stapedectomy with vein graft operation. When speech discrimination losses are considered, however, 1.9 per cent of our vein graft patients suffered severe or disabling permanent hearing losses in the operated ear (see Table I).

In our preoperative examinations routine discrimination tests are performed. Of the 414 patients operated, 382 have had postoperative tests. Postoperative discrimination tests have been performed for 327 of these patients. The significance of postoperative discrimination tests was not noted until 40 vein graft operations had been performed. Since institution of routine postoperative discrimination tests, an

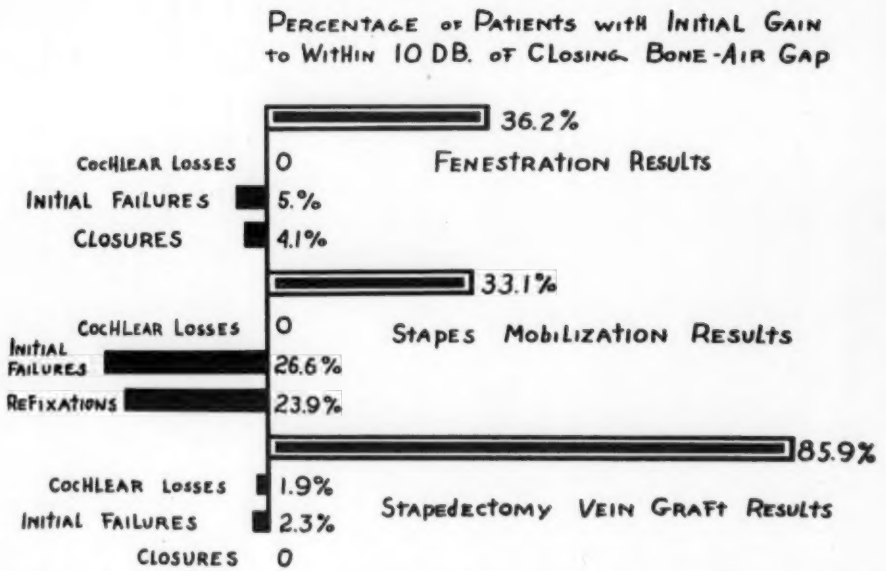

Fig. 1. Comparison of fenestration, stapes mobilization and stapedectomy-vein graft results. Based on 337 fenestrations, 587 stapes mobilizations and 382 stapedectomy-vein graft operations.

additional 15 patients were tested postoperatively elsewhere by pure tone tests only. All who were tested elsewhere, however, and all those who had reported postoperative hearing loss have returned to our office for subsequent discrimination tests. In regard to additional hearing losses after stapedectomy, it is evident that the validity of results based only on pure tone tests is subject to question. The true incidence of the complication of additional postoperative hearing loss (hearing made worse) after stapedectomy may not be evident when tabulation of pure tone tests results only are evaluated; furthermore, the complication of the late hearing loss after stapedectomy may be partially elucidated when routine discrimination tests are done pre- and postoperatively. The only cases of so-called "late losses" which have occurred in our series showed a partial discrimination loss on the one-month postoperative test, which may indicate injury to the cochlea at the time of operation.

TECHNIQUE.

While the method used in this series is essentially the same as that advocated by Shea, some variations in the solution of problems have been developed. In addition, some items of interest related to the operation may be worthy of discussion.

Management of Chorda Tympani Nerve.

The chorda tympani was sectioned in 60 per cent of the cases in this series. If any evidence of trauma—tearing of the nerve sheath, stretching or bruising of the nerve—was evident, the nerve was sectioned. Permanent paresthesia of taste usually develops after injury to the nerve, whereas compensation for the loss of taste after nerve section usually occurs after several weeks. For this reason, all nerves with any evidence of injury have been sectioned.

Removal of Super-structure (Crura) of the Stapes.

Since an intact lenticular process is a vital factor in anchoring the polyethylene prosthesis to the incus, extreme care is exercised in dividing the incudo-stapedial joint. In removal

of the crura the stapes tendon is cut first. The anterior crus is cut as close to the margin of the oval window as is possible with a crural saw-knife. The incudo-stapedial joint is then cut with a knife especially designed for the purpose. The posterior crus is fractured from the footplate by application of firm pressure with a pick, the pressure being applied on the superior side of the crus near the footplate with resultant fracture of the crus against the promontory. Since the crus does not withstand pressure applied in this manner without fracture, it usually shears off at the footplate. This method has been effective in maintaining an adequate lenticular process in every case in which it has been attempted; furthermore, in this series, there has been no instance in which the prosthesis was dislodged from the incus.

Before removal of the crura of the stapes, the mobility of the lateral portion of the ossicular chain (malleus and incus) is routinely assessed. Slight pressure on the under surface of the malleus should result in movement of the malleus and incus. If the lateral chain is not mobile, because of fixation of the malleus, the malleus firmly resists movement on palpation. Fixation of the lateral chain occurred in six instances in this series. On each occasion the anterior surface of the head of the malleus was firmly fused by dense bone to the anterior wall of the epitympanic recess. In five of the six cases, the stapes was also fixed by apparent otosclerosis. In one ear, only the malleus was fixed, and the stapes showed no evidence of fixation. In each instance the history was one of progressive hearing loss throughout adult life. Malleolysis was performed by freeing the head of the malleus with gouges and finishing burrs, after partial removal of a portion of the tympanic plate for proper exposure. After malleolysis, stapedectomy and vein graft with polyethylene prosthesis were performed on the ears in which stapes fixation was a problem. Undetected fixation of the malleus may be the reason for the occasional stapedectomy patient who has insufficient or no postoperative gain. Occasionally insufficient postoperative gain for the low frequencies is noted after stapedectomy, which may also be a result of partial fixation of the lateral chain.

Removal of Mucous Membrane from the Oval Window Niche.

A circumferential incision is made around the oval window niche with a mucous membrane knife. The mucoperiosteum enclosed by the incision is elevated toward the footplate and an attempt is made to remove the elevated membrane in one piece. In histologic studies of the membrane extracted in this manner, gland-like structures are occasionally seen. These may represent invagination of the epithelium. The epithelial cells in these structures are usually tall cuboidal or cylindric in type, and, in some instances, mucicarminophilic material has been demonstrated in the lumina. These gland-like structures have been found in 32 of the 84 specimens studied.

The mucoperiosteum varies in thickness. An active focus of otosclerosis may cause a reaction in adjacent tissues. The membrane that covers a very markedly involved footplate is usually thicker and more adherent to its surface than the membrane that covers an uninvolved footplate.

Control of Hemorrhage.

In addition to routine electrocoagulation of the skin incision margins in the external auditory canal, special measures are applied to the incision line in the mucous membrane in the middle ear. After incision and denudation of the mucous membrane in the oval window niche, small pledgets of synthetic sponge (Curon^{R}) soaked in 1:1000 solution of Adrenal HCL are applied to the mucous membrane margins. The sponge is applied with sufficient pressure to control bleeding completely before any effort is made to remove the footplate. On occasion, it is necessary to apply a small pledget of Gelfoam^{R} to the area first, and then press the Curon on top of the Gelfoam. Removal of the synthetic sponge leaves the small piece of gelfoam pressed against the bleeding point. If the bleeding has been profuse, the gelfoam may be left in place. This method has been entirely effective in controlling mucous membrane bleeding when care is taken to incise the membrane around the complete circumference of the oval window.

Hemorrhage at the time of extraction of the footplate

definitely increases the technical difficulties of the operation and may be associated with a decrease in the efficiency of the hearing result and an increase in the incidence of cochlear losses (see Table II). Since Hohmann has shown experimentally that hemorrhage into the vestibule as an isolated factor does not cause evident damage to the organ of Corti in cats,[3] it is probable that damage associated with hemorrhage in this series is a result of technical difficulties engendered by the hemorrhage and not a result of hemorrhage *per se*. For this reason, the prevention of hemorrhage should be emphasized,

TABLE II.

Results Following Moderate to Marked Hemorrhage Into Vestibule.

1. Results Based on Closure of Bone-Air Gap		
Within 10 db or better	34	77.8%
Within 11-15 db	6	13.3%
Within 16-20 db	1	2.2%
Within 21 db or more	3	6.7%
2. Hearing Made Worse		
Based on Pure Tone Tests	1	2.2%
Based on Discrimination Tests		
Severe Losses	3	6.7%
Partial Losses	1	2.2%
Total Number of Cases	44	

and the removal of blood from the vestibule should be deemphasized. Currently, if hemorrhage occurs after removal of the footplate, no attempt is made to remove the blood from the oval window or the vestibule.

Removal of the Footplate.

After removal of all mucous membrane from the footplate and adjacent wall of the oval window niche, a cut is made across the footplate, perpendicular to the long axis of the window. When the footplate is thin and soft, the cut is made with a fine straight footplate pick. If the footplate is hard or thick, the cross cut is made with a Shea perforating burr. Making the cross cut at this juncture is helpful in avoiding mobilization of the footplate and prevents detachment of the whole footplate that is difficult to extract. Regardless of the thickness of the footplate, a mobilized footplate can be avoided

if this technique is used. If, however, an attempt is made to remove overhanging bone from the niche or to thin the footplate with a finishing burr before the cross cut is made, the entire footplate will be freely mobilized with troublesome frequency. This complication necessitates additional instrumentation such as cutting a notch in the lower border of the window in order to insert a hook under the edge of the footplate. Even though a notch is cut in the inferior margin, a thick footplate may be difficult to extract under these circumstances without rotation of the footplate in the vestibule. Such rotation increases the incidence of trauma to the vestibular contents and cochlea.

After the cross cut has been made, overhanging otosclerotic bone can be removed as desired. Then, chisels are used to mobilize the two fragments. Proper placement of the chisels in the annular ligament area allows for mobilization of large fragments of the footplate. It is not unusual to remove the footplate in two fragments after freeing them in this manner, and removal is not difficult; also, these large fragments afford good material for histological study. Thin fragments are easily removed with hooks after mobilization; however, the removal of markedly thickened totally involved footplates creates the special problems of increased trauma and postoperative closure of the oval window. Shea and Myers have reported an incidence of 3 per cent and 6 per cent postoperative closure, respectively, of the oval window after removal of such footplates.[3,5] This type of footplate has been encountered in 71 instances (19 per cent) in this series. Closure of the window has not occurred in any case to date. Since the method employed results in the total removal of the extremely thickened, totally involved footplate, and subsequent closure has not occurred, the technique will be described in detail. Removal of the footplate is done in a series of definitive steps, after adequate denudation of the mucous membrane.

Step 1. The footplate area is cut across the center with a Shea perforating burr in a line that runs at right angles to the long axis of the oval window. The cut is then widened until a rectangular area (see Fig. 2) is removed from the center of the footplate.

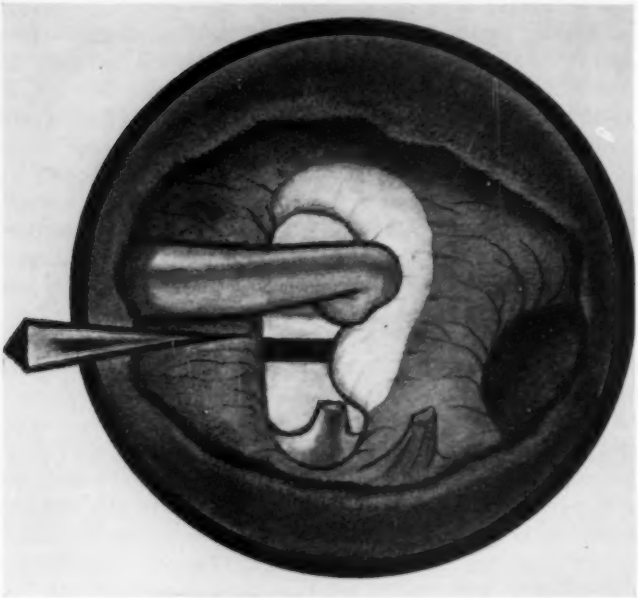

Fig. 2. Cross-cut rectangular opening in footplate after removal of bone with the perforating burr. Mucous membrane has been removed from the walls of the oval window niche and footplate.

Step 2. All overhanging bone is removed from the edge of the oval window niche with a small finishing burr (see Fig. 3). The remaining anterior and posterior areas of the footplate may then be thinned with the finishing burr if necessary. After removal of the overhanging bone and thinning of the footplate fragments, the annular ligament area is usually seen as a fine line of demarcation.

Step 3. Chisels are next used to mobilize the anterior and posterior fragments (see Fig. 4). On mobilization the fragments settle slightly in the niche, especially in the area of the cross cut made in Step 1. If the cross cut is of insufficient width, the fragments will come together as they become depressed. In Step 1 the cross cut should be made of sufficient width in the form of a definite rectangle in order

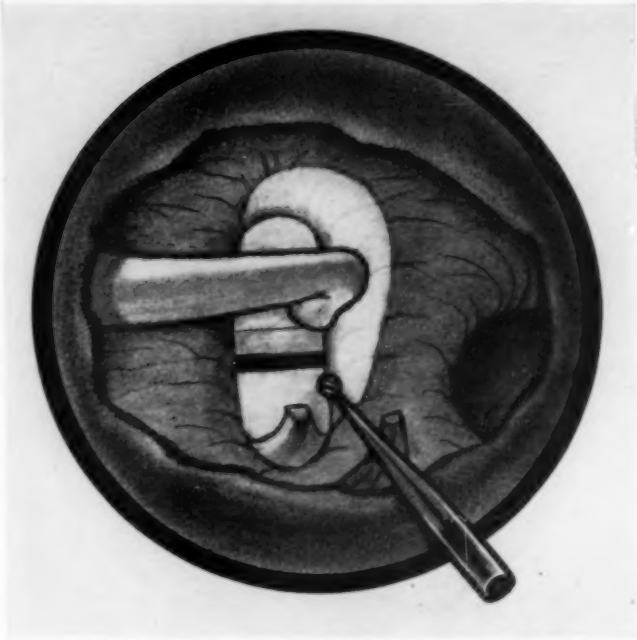

Fig. 3. Overhanging bone removed with finishing burr.

to maintain an opening between the fragments after they are mobilized. Insufficient width in the cross cut area in the anterior-posterior diameter makes removal of the fragments difficult. Before proceeding to the next step, the edges of the niche should be inspected again for overhanging bone. If there are overhanging edges, they should be removed with the finishing burr with the mobilized fragments in place.

Step 4. A right angled footplate hook is then inserted through the cross cut area, and the anterior and posterior fragments are rolled out of the niche (see Fig. 5). Clean removal of the footplate and a clean oval window are thus attained. In the event of hemorrhage from the annular ligament area on partial removal of the first fragment, the fragment can be returned to its original position in the window

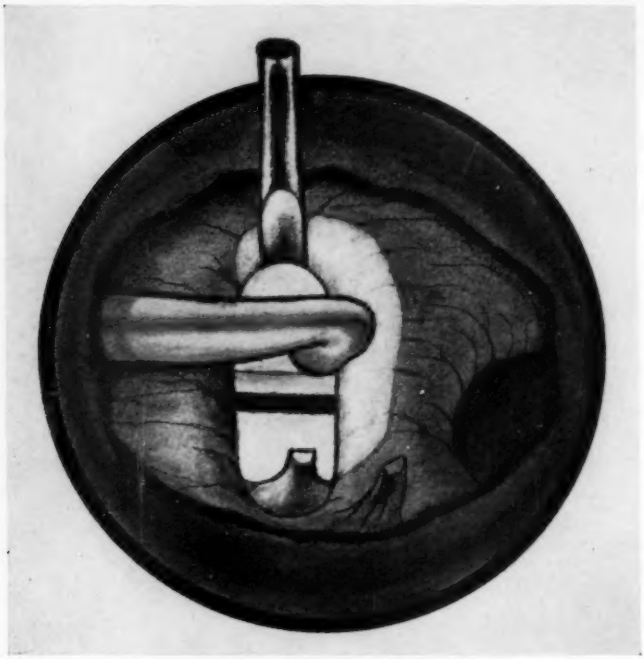

Fig. 4. Use of chisels to mobilize fragments.

until the hemorrhage is controlled. Should hemorrhage occur on removal of the second fragment, the vein graft can be promptly placed in the oval window.

The incidence of severe cochlear losses after removal of the markedly involved footplate has been 4.2 per cent as compared to 1.9 per cent in the overall series, and the hearing efficiency has been slightly decreased (see Table III). The judicious use of chisels does not appear to be a factor in the incidence of marked cochlear losses, since such losses are as frequent in cases in which the chisel was not used as in those in which the chisel was used. In addition, the complication of increased cochlear loss did not occur in 116 cases in which the chisel was used in our mobilization series. In the last 25

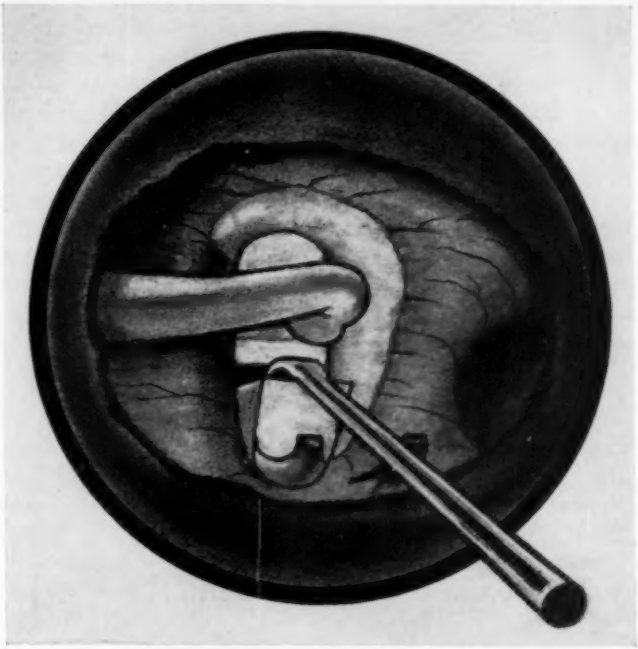

Fig. 5. Removal of footplate fragments from oval window with hook.

patients there has been no incidence of cochlear losses after removal of this type of footplate. Improved technical skills will probably further reduce the incidence of cochlear losses in these difficult cases. A total of 33.9 per cent of the footplates were involved with a gross otosclerotic focus which encompassed at least half of the footplate area. In 46.8 per cent of the patients, there was minimal gross otosclerosis with only the margin of the footplate being involved.

Saucerization of the Oval Window Niche.

Markedly overhanging bone on the promontory which would obstruct removal of the footplate or proper position of the prosthesis or the vein graft is removed. Routine saucerization

or removal of small foci of otosclerosis on the promontory has not been performed.

The Vein Graft.

A moderate size (4 x 5 mm.), moderately thick to thick graft has been used in 268 of the 382 ears operated upon in this series. The thickness of the graft is governed by the thickness of the substantia propria and the amount of adventitia which remains on the graft. The graft is tailored to conform to the niche in such a manner that it will drape into the con-

TABLE III.

Results Following Removal of Totally Involved, Solid Footplates.

1. Results Based on Closure of Bone-Air Gap		
Within 10 db or better	57	80.3%
Within 11-15 db	8	11.3%
Within 16-20 db	2	2.8%
Within 21 db or more	4	5.6%
2. Hearing Made Worse		
Based on Pure Tone Tests	1	1.4%
Based on Discrimination Tests		
Severe Losses	3	4.2%
Partial Losses	0	
Total Number of Cases	71	

cavity of the niche as it clings to its walls and the inferior edge of the facial nerve. The graft is not large enough to drape over the lateral surface of the nerve. When a vein with thick substantia propria is used in a moderate-sized niche, the adventitia is trimmed completely away from the vein; however, when the substantia propria is thin, a moderate amount of adventitia is left on the graft so that when it is placed in a wide niche a proper volume of tissue will be provided to give good support to the prosthesis. Occasionally, when the vein available has a thick substantia propria, it is necessary to enlarge a narrow niche by saucerization in order to accommodate the volume of tissue.

After removal of the appropriate amount of adventitia, the vein is opened longitudinally, and with the intimal surface directed downward, is placed on a tongue blade. The graft is then cut to the desired size and shape by the use of a sharp

plastic-type knife blade. Care is taken to avoid inclusion of exceedingly thin or perforated areas in the graft, since adequate strength of the graft for support of the prosthesis and possible resistance to erosion from the end of the prosthesis may be important factors in maintenance of maximum hearing levels over long periods. For example, in one patient, the end of the prosthesis herniated through the substantia propria of the graft. The patient subsequently had an appreciable loss of hearing efficiency, both a diminished acuity for pure tones as well as discrimination loss. Later exploration and removal of the prosthesis showed that it had herniated through the vein. Although the prosthesis had maintained its attachment to the lenticular process, its lower end had protruded through the substance of the vein and was covered by a thin membrane which formed a small blister-like dome on the surface of the new oval window membrane upon removal of the prosthesis. The graft was removed and replaced with the result that hearing was restored to the former adequate postoperative level for pure tone and discrimination. Sections through the herniated area of the removed graft showed an extremely thin area of loose connective tissue covered by mucous membrane with thicker, more compact connective tissue on either side.

Although Shea advocates the use of a vein press to insure a thin graft,[9] evaluation of the postoperative hearing results in this series indicates that the thickness of the graft had very little influence on the hearing results (see Fig. 6). Five of the seven patients who had severe cochlear losses in the series had marked preoperative evidence of nerve degeneration as judged by audiological findings; hence, the slightly increased percentage of cochlear loss which appears to be associated with the use of thick grafts is believed to be the result of multiple factors rather than of thickness of the graft alone.

The Prosthesis.

Animal-tested polyethylene tubing taken directly from the electron sterilized package (Intramedic^{R}) was used in all cases. Gauge #90 tubing, made slightly expandable at the

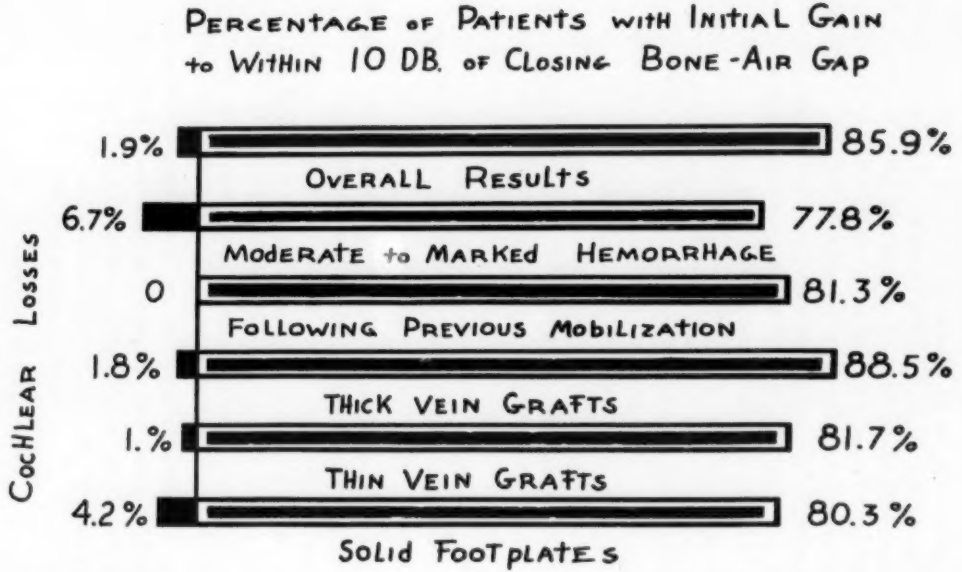

Fig. 6. Comparison of hearing results and complications when moderate to marked amounts of hemorrhage into the vestibule was encountered (45 patients), following previous mobilization (80 patients), use of thick vein grafts (268 patients), use of thin vein grafts (98 patients) and removal of solid footplates (71 patients) as compared to overall stapedectomy-vein graft results (382 patients).

upper end by several longitudinal cuts around the circumference, was used principally. Gauge #50 tubing was used in one instance of a very small lenticular process, and #160 tubing in two patients with large processes. The length of the prosthesis varied from 3 to 5 mm., with the prosthesis being 4 mm. long in 82 per cent of cases. A 3 mm. prosthesis was used in two patients, and one 5 mm. was used in three patients.

The prosthesis should be long enough to project slightly into the vestibule to insure stability of position. Each prosthesis was fashioned in shape and length to fit the individual requirements of the anatomy of the incus and oval window niche.

In placing the prosthesis, the tip is adjusted into the central zone of the graft with a pick. Then by use of bimanual manipulation with picks, the incus is slightly elevated and the prosthesis moved into position with the lenticular process extending into the upper end of the prosthesis. A minimum of adjustment of the graft is necessary after positioning of the prosthesis.

POSTOPERATIVE COURSE.

The surgical dressings are removed on the second postoperative day. This includes removal of the external auditory canal pack—the cigarette paper strips over the incision line, and the cotton balls tied together with silk sutures.

Antibiotic Medication.

Comycin[R], 500 mgm. every eight hours is administered preoperatively, and for 36 hours postoperatively while the patient is in the hospital. After dismissal, the patient continues to take 250 mgm. of Comycin every eight hours for a period of four days. In addition, Terfonyl[R], 1 gm. is given postoperatively for a period of six days.

Vertigo.

Mild to moderate vertigo occurred in 63.2 per cent of patients. Vertiginous symptoms were reported by 24.5 per

cent of patients for a period of less than one week, by 25.2 per cent for one to two weeks, and by 13.5 per cent for two to three weeks.

Hemotympanum.

Ninety-six patients had a definite hemotympanum as seen on postoperative examination of the tympanic membrane. The clot in the middle ear was associated with a poor hearing gain immediately after operation, and there was no improvement 12 to 14 days before absorption or drainage of the clot occurred spontaneously. During this period distortion of hearing and poor discrimination were invariably present. The hemotympanum did not affect the final hearing result, how-

TABLE IV.

Changes in 4000 and 8000 Frequencies.

	4000 Frequency	8000 Frequency
Results Based on at Least 10 Db Gain or Loss		
Gain	300—77.8%	169—43.9%
Loss	27— 7.1%	69—17.9%
Unchanged	58—15.1%	147—38.2%
Total Number of Cases	385	

ever, since 88.5 per cent of the ears with hemotympanum gained within 10 db. or more of closing the bone-air gap. Two per cent of the hemotympanum group had marked cochlear losses as compared to 1.8 per cent cochlear losses in the group with no hemotympanum.

Postoperative Loss of High-tone Perception.

There has been much speculation about the incidence of permanent hearing losses for the 4000 and 8000 frequencies after the stapedectomy and vein graft procedure. The changes in this series in the 4000 and 8000 frequencies, based on the most recent tests, are given (see Table IV). The gain in these frequencies is usually not complete at the time of the one-month postoperative test, and the final level is usually attained between the first- and fourth-month tests.

Duration of Results.

Table V illustrates the stability of the hearing gain as tabulated by the one-month, 12-month, and 18-month tests. Patients who achieved good discrimination, as shown in the one-month postoperative test, have uniformly maintained it. There have been no closures of the oval window to date.

DISCUSSION.

On the basis of present evidence the Shea operation is a relatively safe procedure when meticulously performed on the typical patient with stapes fixation due to otosclerosis.

TABLE V.

Stability of Postoperative Results.

	Total Operations	With Tests	Gain to Within 10 Db or Better	Severe Cochlear Losses	Post-operative Closures
One Month (or first test)	414	382	319—82.9%	7—1.9%	0
One Year	204	129	113—87.5%		0
18 Months	97	73	62—84.9%		0

Removal of a markedly involved footplate and poorly controlled hemorrhage at the time of its extraction are factors which may adversely affect the postoperative hearing result. This adverse effect probably results from the increased technical difficulties involved during the operation rather than to the hemorrhage itself. Neither of these problems is insurmountable, and in view of the generally good results obtained, there should be only slight concern regarding future acceptance of this method.

Unfortunately, all too often the innovation of a surgical technique is discredited before proper evaluation has been made on the basis of data obtained under controlled conditions. Because of past experience regarding judgment of a new surgical procedure, carefully planned and thoroughly executed preoperative and postoperative examination, as well as skillfully performed surgery, is admittedly the *sine qua non* of ultimate scientific evaluation. Since the early reports of

results of the Shea operation by various authors have been extremely encouraging, it is hoped that this operation will be assessed in its proper perspective. Although this technique must be carried out with care, it can also be done with confidence that in the majority of cases, satisfactory hearing results will be obtained; indeed, the technical problems that exist can undoubtedly be solved by the recognition of faults in technique and by improvement of technical skills. I am confident that further research on these attendant problems will reduce or ultimately eradicate the complication of additional cochlear loss in the meticulously-performed stapedectomy of the future.

SUMMARY.

A resume of experience with the Shea oval window-vein graft technique used in 414 operations has been presented. Improvement in hearing was evident with regard to closure of the bone-air gap, initial failure, and refixation and closure of the fenestra. From assessment of pure tone loss only 0.7 per cent of patients showed increased hearing loss, whereas assessment of speech discrimination loss showed 1.9 per cent of patients with severe hearing losses in the operated ear. It has been pointed out, however, that when routine discrimination tests were done postoperatively, only the cases of so-called "late losses" showed a partial discrimination loss on the one-month test. This has been construed as possible evidence of preexistent cochlear injury.

Personal modifications used in the operation have been described, and a detailed step-wise procedure for removal of the footplate has been given. It is believed that damage associated with hemorrhage in this series was a result of technical difficulties engendered by the hemorrhage and was not a result of hemorrhage *per se*.

In general, patients who showed good discrimination on the one-month postoperative test have maintained it, and there has been no instance of closure of the oval window after operation. On the basis of results obtained in this series, the

Shea operation is recommended as a safe and effective means of restoring hearing in patients with otosclerosis.

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RESULTS WITH THE POLYETHYLENE T-STRUT IN THE RESTORATION OF HEARING.*

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In an attempt to improve the level of hearing in a tympanoplasty procedure, such as a Wullstein Type IV, where the only remaining portion of the ossicular chain is a mobile footplate of the stapes, various prostheses have been used. Wullstein[1] reported the use of an oval ring of "polavit" made to fit in the oval window niche between the mobile footplate and the tympanic graft, acting as an acoustic transmitter. Shambaugh[2] and Guilford[3] have reported improved results in Type IV tympanoplasty with the use of a polyethylene strut between the footplate and the tympanic graft; however, this type of prosthesis in most cases has mainly failed to maintain good improvement of hearing, and the most reliable method for handling Type IV tympanoplasty has been to adjust the tympanic graft so that the round window is protected and the oval window is exposed to the sound waves. In my experience the hearing improvement obtained with this type of reconstruction in most cases was not good; and, therefore, another type of prosthesis was given a trial.

In trying to use a single strut I found it quite difficult to maintain its correct position, and an effort was made to create a more stable prosthesis. A cross bar polyethylene prosthesis was tried in several cases.[4] This consisted of a No. 50 polyethylene strut with one end flared widely and the other end slightly tapered, going loosely through a piece of No. 205 polyethylene tubing (see Fig. 1), the No. 205 tubing helping to maintain the No. 50 strut in its proper position. This prosthesis was found quite difficult to place in proper

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position in some cases because of the angulation of the facial nerve with the oval window. In selected cases, the mobile footplate was removed and the oval window covered with a vein graft or flattened gelfoam (see Fig. 2), thus giving the strut a better "footing," particularly where the oval niche was shallow.

Case 1. Sept. 30, 1959. A radical mastoidectomy was performed, and a skin graft was used for reconstruction of the tympanic membrane and middle ear space. The footplate was not mobile and was removed. A vein

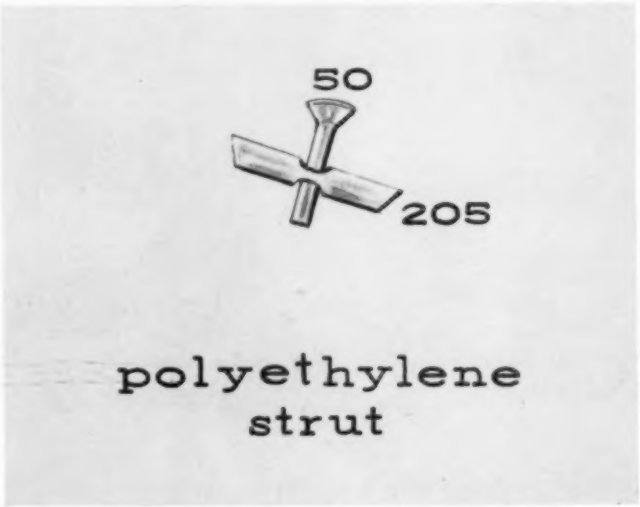

Fig. 1.

graft was used to cover the oval window and a cross bar prosthesis was inserted between the oval window and the tympanic graft.

The strut extruded after eleven months but the hearing improvement remained. Adhesions must have formed between the new tympanic membrane and the oval window after extrusion of the strut.

Case 2. Nov. 2, 1959. A radical mastoidectomy was performed. The suprastructure of the stapes had been destroyed but the footplate was 100 per cent otosclerotic and immobile. The footplate was removed, and the middle ear was reconstructed with the help of a skin graft and cross bar prosthesis. Flattened gelfoam was used to cover the oval window.

This patient has continued to maintain his hearing improvement except for fluctuations due to secretory otitis media which he has bilaterally.

He now has a polyethylene tube through the graft into the middle ear and is watched closely.

Case 3. Dec. 30, 1959. A radical mastoidectomy was performed. A mobile footplate in a rather deep niche was left intact. After reconstruction of the middle ear with a skin graft, a cross bar prosthesis was inserted.

The No. 50 strut was extruded five weeks postoperatively. The hearing improved as shown by the audiogram three weeks postoperatively, only to drop back to its original level after extrusion of the No. 50 strut.

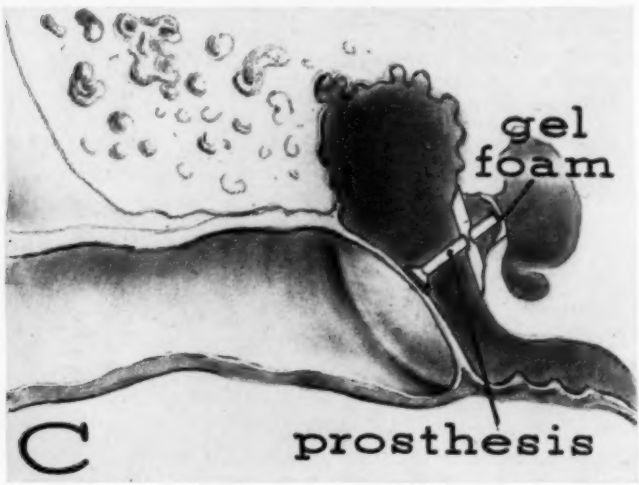

Fig. 2.

Case 4. Jan. 27, 1960. A Type IV tympanoplasty with antral control opening only was done. The middle ear was reconstructed with the use of a full-thickness skin graft. A vein graft was used for a denuded mucosal area in the hypotympanum and for protection of the oval window after the footplate of the stapes was removed. The strut extruded after five months following an initial period of good improvement of hearing.

Case 5. Jan. 29, 1960. A fenestration of the horizontal canal was performed on the right ear in 1954 and on the left ear in 1955. The hearing improvement was maintained in the right but receded after four months in the left. She did not wish to have a revision of the original fenestration operation and was not a good candidate for it; therefore, a stapedectomy was performed, gelfoam was used to cover the oval window, and a cross bar prosthesis was inserted. Good improvement of hearing was maintained six months, only to have the No. 50 strut extrude.

She returned in two weeks, and the perforation was healed where the strut had extruded. The initial hearing improvement was present. Adhesions must have developed between the drum and oval window.

Case 6. In June 1959, a tympanoplasty Type III was performed with the use of a skin graft attached to the head of the stapes. In March 1960, the middle ear was examined again because of failure of any hearing improvement. The stapes was found to be immobile because of tympanosclerosis filling the oval window niche. A stapedectomy was performed and gelfoam used to cover the oval window. A cross bar prosthesis was inserted and the graft replaced. Good hearing improvement was maintained for nine months, only to have both parts of the prosthesis extrude with a drop of hearing to the original loss.

Case 7. April 2, 1960, a radical mastoidectomy was required. The only ossicular chain remnant was a mobile footplate situated in a deep oval

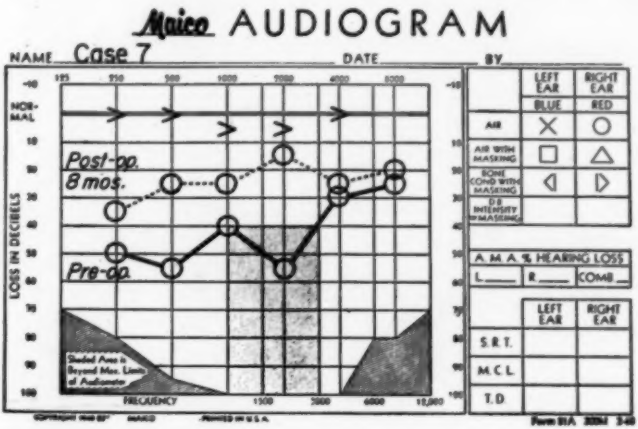

Fig. 3.

window niche. A cross bar prosthesis was inserted between the full-thickness tympanic graft and the stapes footplate. Good improvement of hearing has been maintained so far (see Fig. 3).

After using the cross bar prosthesis in several other similar cases, I decided to modify it because of its tendency to extrude and the difficulty of correct placement. A single No. 90 strut with a wide flared brim was used with a piece of vein anchored to the bottom by stainless steel wire looped around the vein and both ends of the wire run through the strut and out beneath the brim of the strut (see Fig. 4), the idea being that after removal of the footplate, the vein plug would close the oval window and at the same time by its attachment to the oval window niche would keep the strut from extruding.

Case 8. Aug. 9, 1960, a fenestration of the right horizontal canal was performed. Good improvement of hearing was maintained for four months, then the fenestra closed. In June 1959, a fenestration of the oval window was performed with maintenance of good hearing. She did not wish to have a revision of the fenestration of the horizontal canal performed. In August 1960, the middle ear was exposed, and the angulation of oval window and malleus would not permit a strut from the malleus to the oval window to be used. A stapedectomy was completed, and a polyethylene strut with a small piece of vein attached by stainless steel wire was inserted. Good improvement has maintained itself for five months.

Case 9. Oct. 11, 1960, a fourteen-year-old boy with a conductive loss of hearing for four years or longer was examined. There was no history

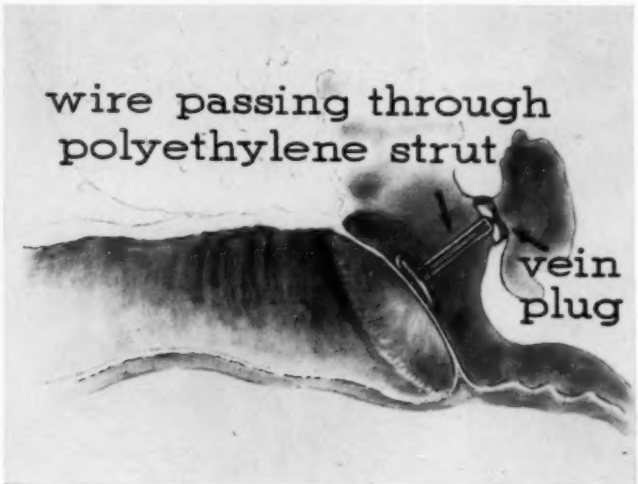

Fig. 4.

of middle ear infection, and the tympanic membrane appeared normal. X-rays of the mastoid showed some antral sclerosis in the right mastoid. The right middle ear was explored only to find a marked enlargement of the facial nerve. This swelling of the nerve had caused erosion of the long process of the incus and complete destruction of the suprastructure of the stapes. The oval window niche was four-fifths obliterated by the nerve (see Fig. 5). Since there had been no evidence of seventh nerve disturbance prior, nothing was done to the facial nerve. A polyethylene strut with a vein attached was slipped into the oval window after removal of the footplate. So far, moderate hearing improvement has been maintained.

Case 10. Dec. 30, 1959, a tympanoplasty was performed with the use of a cross bar strut which extruded after five weeks, but the graft remained intact. In November 1960, the middle ear was exposed and the

mobile footplate of the stapes removed. A polyethylene strut with a vein attached was inserted in the oval window; also, an additional vein graft was used beneath the graft and brim of the strut as further reinforcement of the drum. So far, good improvement of hearing has been maintained for two months (see Fig. 6).

CONCLUSION.

The cross bar polyethylene prosthesis was found not to be satisfactory for use as an acoustic transmitter in the majority

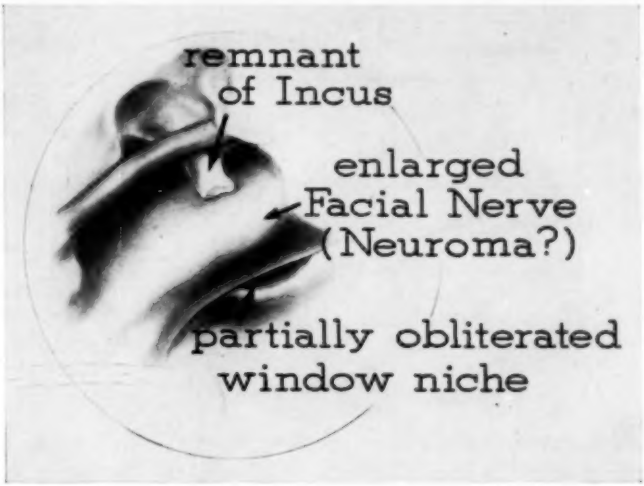

Fig. 5.

of the cases presented. Correct placement of the prosthesis was difficult in most cases because of angulation of the facial nerve with the oval window niche. In five out of the first seven cases presented, after initial good hearing improvement extrusion occurred even as long as eight to ten months later. The prosthesis that has remained the longest with good hearing improvement remained in position for thirteen months. Luckily, two of the cases with extrusion of the strut were followed by good hearing improvement after adhesions appar-

ently had developed between the tympanic membrane and oval window, with the footplate of the stapes having been removed.

The flared strut with the vein plug wired to its end was found much more practical to use and seems to offer a better chance for permanent transmission of sound than the cross bar prosthesis; however, the oldest of these is six months.

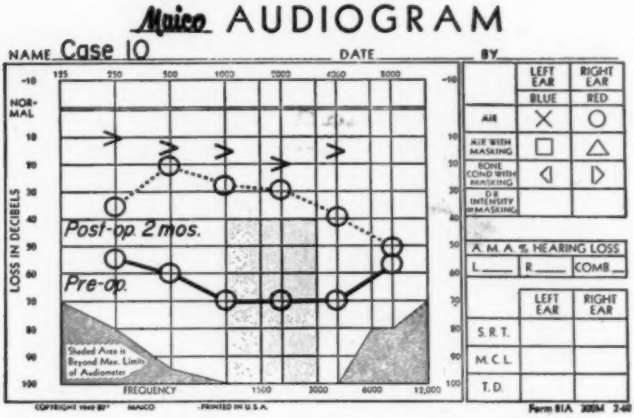

Fig. 6.

At the present time, in any case of Type IV tympanoplasty which I think is suitable for a prosthesis, the following approach is used: After removal of all the pathology and reconstruction of the middle ear and tympanic membrane has been completed, a very short cross bar prosthesis with a broad flared brim is inserted between the footplate and tympanic graft. This prosthesis may serve in an occasional case as a satisfactory columnella and in most cases helps preserve the middle ear space; if it extrudes, the perforation is found to close quickly. After healing is complete, and the hearing level is not satisfactory—or if the hearing was improved, but the prosthesis was extruded with a persistent decrease in hearing —a polyethylene strut with a vein plug attached and removal of the footplate is used as a secondary procedure. The tym-

panic graft may be reinforced with a vein graft between it and the flared end of the strut.

This same strut with a vein plug attached may be worthwhile in revising a fenestration of the horizontal canal if the angle between the oval window niche and the malle s not lend itself readily to the use of a polyethylene to the use of a fat plug attached to the malleus with a st steel wire.

Removal of the footplate and insertion of a polyethylene strut with a vein plug attached is performed as a secondary procedure.

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EIGHTH ARGENTINE CONGRESS OF BRONCHOESOPHAGOLOGY.

The Eighth Argentine Congress of Bronchoesophagology will be dedicated to the memory of the late Chevalier L. Jackson, M.D., of Philadelphia, Penna. This Congress will be held in Tandil, province of Buenos Aires, Argentina, November 17-20, 1961, under the presidency of Dr. Juan Carlos Arauz, assisted by several foreign physicians. For further information write Dr. Juan Blank, Secretary Cangallo 2150 BsAs, Buenos Aires, Argentina, South America.

FOOD ALLERGY IN THE EAR, NOSE AND THROAT PRACTICE OF ALLERGY.*

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Regardless of whether or not he is engaged in the active practice of allergy, the present day otolaryngologist must be aware of allergic reactions in man, for he is vitally concerned with the primary shock organs involved in these reactions. Whereas much is known and documented regarding the effect inhalent allergens have upon the respiratory system, the converse is true as regards our knowledge of the effects ingestant allergens have upon the digestive and metabolic system of man, particularly the oropharynx, hypopharynx and esophagus which may logically be considered as the primary shock organs involved. One of the main reasons for this situation is our lack of knowledge concerning the cause and effect relationship between the ingestion of food and the causation of symptoms, with the consequent failure to recognize these symptoms when they occur.

The purpose of this paper is to correlate modern concepts and ideas of research scientists working in the broad field of hypersensitivity, with observations made upon patients suffering from food allergy, in an attempt to explain the cause and effect relationship between the ingestion of foods and the causation of symptoms in man. In doing so, we are fully cognizant of the warning that what is observed in the experimental animal under controlled laboratory conditions does not always apply to man. Our reasoning is based upon theories and facts presented by research specialists in the two most comprehensive symposia on the subject of hypersensitivity to be found in the literature to date.[1,2]

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BASIC CONCEPTS OF ALLERGY.

Allergy can be regarded as an aberration of the normal defense mechanism of the body. It is an altered reactivity from the normal, or average response of an organism to the entrance of a foreign substance or to external stimuli and is considered to be an antigen-antibody reaction even though antibodies are not always demonstrable. The presence of skin sensitizing antibodies as illustrated in the passive transfer phenomenon can be better understood when one realizes that the skin's chief function is to protect the body it encloses. Antigen antibody reactions which are beneficial to the organism are designated as immune reactions, while those which prove harmful are considered as examples of hypersensitivity, yet the basic mechanism of antigen antibody combination in immunity and hypersensitivity are similar.

CLASSIFICATION OF ALLERGIES.

Allergic reactions may be classified on the basis of the rapidity with which they appear. In the immediate or anaphylactic type the reaction occurs within minutes after exposure to a foreign substance. It may last for an hour or more, then gradually subsides. The classical example is the wheal which occurs when a skin test is positive to an injected antigen, such as an extract of pollen or house dust. The response observed clinically is dependent upon the shock organ or tissue involved. Asthma, hay fever, coryza or urticaria are illustrative of such responses. The immediate reaction is primarily a vascular phenomenon caused by an increase in the permeability of blood vessel walls, due to the release of substances resulting from the union of antigen and antibody. The antibody involved had been formed as a result of previous invasion of the organism by a foreign substance, which then penetrated body cells and participated in the metabolism of these cells, producing antibodies which were released in body fluids where they circulated freely. When specific available antigen is encountered pharmacological substances are released which cause alteration in the endothelial lining of blood vessel walls. Cellular tissue damage *per se* does not

occur except from the adverse effects of impaired circulation, as demonstrated in the Arthus' phenomenon. This type of reaction is characterized by focal eosinophilia, the presence of circulating antibodies as demonstrated by the passive transfer test and the regression of symptoms following injection of epinephrine.

In the other form of allergic response designated as delayed hypersensitivity or delayed allergy, the reaction may not appear for hours, will reach its peak in about two days, then gradually subside in days or weeks. The reaction elicited is acutely inflammatory in nature with round cell infiltration and other evidences of a phlogistic response. The tuberculin skin test is the best example of delayed hypersensitivity and is frequently utilized in experimental work. Certain bacteria, fungi, viruses and contact with allergenic substances such as poison ivy, poison oak or sumac can elicit a delayed response. It is felt that the reaction may be related to the chemical complexity of the antigen involved. Prolonged contact or repeated applications of the allergenic substances involved may produce an inflammatory response in the skin characterized by erythematous inflammatory papules which may go on to vesiculation and scabbing. Significant features of the delayed reaction are the absence of any demonstrable serum antibody level, focal eosinophilia is lacking, and injected epinephrine elicits no response.

In a study of the delayed hypersensitivity reaction, Lawrence[3] developed a technique whereby tuberculin sensitivity is transferred in man by means of leukocytes. He also demonstrated that extracts of sensitive cells (leukocytes) were as effective as viable cells. Lawrence postulates that a "transfer factor" is involved which is not an antibody of the conventional type. The proof that antibody-modified polymorphonuclear cells can be transferred into a non-sensitized subject, quantitatively demonstrates a difference from the serum antibodies involved in the immediate reaction.

In many respects immediate hypersensitivities are similar to delayed hypersensitivities yet in some significant respects they are different. The basic mechanism of antigen antibody

combination in both types of reaction is similar. In the immediate allergic response, antibodies which had formed following previous exposure to a specific antigen, circulate freely in body fluids and combine with available antigen to release a substance which affects the endothelial lining of vascular spaces, and a transudate results with no accompanying tissue damage. In the delayed reaction, the antibody becomes intimately bound to body cells and does not appear in the circulation as a freely floating substance. When available antigen reaches the fixed antibody, the body cell becomes damaged and an inflammatory reaction with round cell infiltration results. As a rule, the delayed type of reaction requires long contact with the offending agent. Unlike other immune responses mediated by serum antibody, the manifestations and transfer of delayed allergy are intimately bound to the cells of the sensitive host.

Delayed hypersensitivity plays an important role in the host's response to viral, fungal and bacterial infections which are characterized by a chronic course and granulomatous inflammatory response. Blocking tissue antibodies may play a major part in the basic immunological mechanisms involved. Many hitherto obscure diseases such as the collagen diseases[3] may have a common basic etiology with allergic reactions.

MANIFESTATIONS OF DELAYED ALLERGIC REACTIONS TO FOOD.

In a clinical evaluation of a group of patients with proven food allergy,[4] we were intrigued by the high percentage of oropharyngeal, hypopharyngeal and laryngeal symptoms and physical findings which could be attributed to the ingestion of foods to which the patients were allergic. As our awareness of the significance of these signs and symptoms increased, we have subsequently become more impressed with our findings in other food allergic individuals. Certain characteristics of food allergy which we observed, coupled with reports in the literature by other authors, have led us to the conclusion that manifestations of food allergy observed by the otolaryngologist are due not only to the immediate allergic reactions to food, but also to allergic reactions of the delayed type which play an equal, if not a more important role in the

production of signs and symptoms due to food sensitivity. Immediate reactions to food such as acute rhinorrhea, asthma, urticaria and angio edema are well recognized and in fact are the basis of food ingestion tests;[5] yet many clinical and laboratory findings associated with food allergy are difficult to explain on the basis of an immediate allergic response and require a more adequate explanation before the cause and effect relationship between the ingestion of foods and the production of symptoms can be established. In the following paragraphs we will enumerate clinical and laboratory findings associated with food allergy observed in man and will point out features which substantiate our belief that the ingestion of food can produce reactions of the delayed type in man.

THE CYCLIC NATURE OF FOOD ALLERGY AND THE PHENOMENON OF MASKING.

Rinkel's outstanding contribution to our knowledge of food allergy has been the description of the cyclic nature of food allergy and the phenomenon of masking.[5] He has shown that in most instances of specific food sensitization, the food must be eaten during a definite period of time prior to the production of symptoms, indicating that prolonged exposure to the offending allergen is necessary before symptoms become manifest, just as in contact dermatitis, which is considered an example of delayed hypersensitivity. In what portion of the digestive tract this occurs, how absorption of the allergenic factors in food is affected and what stage in the breakdown of the protein molecule is involved is not known[6]; likewise, the phenomenon of masking indicates that the products of food metabolism and assimilation are present in the body over prolonged periods of time, as measured in hours or days and could produce symptoms of delayed hypersensitivity.

THE ROLE OF SKIN AND MUCOUS MEMBRANE IN DELAYED HYPERSENSITIVITY.

Pappenheimer and associates[7] stress the importance that skin plays in the development of delayed hypersensitivity. Delayed reactions to rabbit and guinea pig serum were pro-

duced in persons who had received repeated intradermal injections of these antigens. Studies of sensitivity to simple chemicals likewise revealed the importance of the intradermal route. Topical application was more effective than parenteral injection. They feel that perhaps the proteins of the skin which conjugate with these simple reactive chemicals play a role comparable to that of antibody. The importance of the intradermal route suggests that introduction of a protein in the form of a large complex with antibody causes antigen to be retained in the skin and thereby facilitates sensitization.

Skin tests to food extracts are generally considered to be unreliable by most allergists. This has been our experience. The implication is that skin has not developed through the process of evolution to protect the organism against harmful foods, as it has against foreign substances such as bacteria and simple chemicals. It is reasonable to apply Pappenheimer's theories regarding the importance of skin in the development of delayed hypersensitivities to the role that mucous membrane of the oral cavity and possibly the entire gastrointestinal tract may play as the prime mediator in the production of delayed hypersensitivity response to exposure with foods to which the individual is allergic. Protein in the mucous membrane could conjugate with the large protein complex of a given food and produce an antigen antibody response of the delayed type. If this assumption is correct, oral mucous membrane tests to foods should be more reliable than skin tests. Such tests have been tried and were found to be of value in detecting certain foods suspected of causing canker sores.[8] The squamous epithelial lining of the oral cavity likewise indicates a similarity in function on a histologic basis. Clinical findings will be presented which suggest that the oral mucous membrane has such a function.

OROPHARYNGEAL, HYPOPHARYNGEAL AND LARYNGEAL MANIFESTATIONS OF DELAYED HYPERSENSITIVITY.

The most striking manifestations of delayed hypersensitivity reactions to foods can be observed in the oropharynx,

hypopharynx and larynx because the mucous membrane lining of these areas is the primary shock organ to come in contact with the ingested allergen. The period of contact may be relatively brief, but repeated contact, as in the case of milk, is no doubt a factor. Likewise the food remains in the body for hours thus providing a reservoir of antigens which can be absorbed from the intestinal tract and reach the mucosa via the circulation. In the skin, to cite an analogous situation, prolonged direct contact with poison ivy is not necessary for the development of contact dermatitis. We feel that the esophagus is also involved. Here the opportunity for direct observation is limited because the performance of routine esophagoscopies in food allergic individuals is not warranted. In a few instances we did perform esophagoscopies in food allergic patients for other reasons. The findings were highly suggestive of a delayed type of reaction in the esophageal mucosa of two such individuals.

Most of the patients with throat symptoms which could be attributed to foods complained of a dull, chronic, irritated feeling in the throat which persisted during most of the day. When asked to localize a point of greatest discomfort, they would generally reply that "it's all over the throat." At times the pain would be accentuated and become dull and throbbing in character. At times the mucosa was inflamed and tenderness could be elicited along the sides of the neck. They often had a slight hacking cough and cleared their throats at frequent intervals, yet the mucous drainage raised was scant. They frequently were awakened at night because of a dry, irritated throat which ached and was uncomfortable, and gargling gave little relief. At holiday times when dietary indiscretions were common, they noted a definite accentuation of symptoms. The more astute observers were definite in asserting that ingestion of certain foods made their throat symptoms worse. If examined at this time, the mucous membrane showed accentuation of the inflammatory changes. Associated complaints of burning in the mouth, itching of the palate and a tingling sensation along the tongue margins were not uncommon.

The findings upon physical examination were typical. The

mucous membrane of the oropharynx and hypopharynx was dull red in appearance with small islands of lymphoid hyperplasia throughout, giving the surface a granular appearance. Frequently the lateral pharyngeal bands were hypertrophied from the region of the fossa of Rosenmüller into the hypopharynx. At times the surface would be coated with a fine tenacious film of mucus which was glue-like in character. Smears taken for cytologic study usually failed to show eosinophilia. At times the uvula would be thickened and edematous. The faucial tonsils were not enlarged unless a supervening acute tonsillitis attack occurred. They, too, had an appearance suggestive of chronic inflammation. The tongue appeared smooth and glossy, but at times shallow fissures would appear. In the younger age group the mucous membrane had more tendency to be edematous than in older individuals. An associated lymphadenitis was noted in most patients.

The clinical picture was that of a chronic inflammatory reaction, yet response to antibacterial agents was lacking. A common history given by patients with this affliction who were referred to us by physicians, or came of their own accord, was that they had repeated courses of antibiotic therapy with no relief of symptoms. Tonsillectomies were often done, but this procedure seemed to make the symptoms worse.

These patients responded best to a therapeutic regime of mild astringent gargles followed by an antihistaminic drug in liquid form, which was held in the mouth for its topical effect. Whether the relief obtained was due to an inherent local anesthetic action of the drug, or to an antihistaminic effect, is not known. The relief of symptoms, the improvement in the appearance of the throat following the detection, and elimination of the offending food was gratifying to the clinician as well as to the patient. The most common foods encountered were those most frequently present in the patient's diet. Wheat, corn, milk, egg, beef, pork, tomato, orange, potato and coffee were the most important.

The greatest difficulty encountered was in detecting and

eliminating the foods. Skin tests in themselves were of little value. They had to be supplemented with careful ingestion tests. We feel that the mucous membrane of the oropharynx would be a more logical testing site for foods than is the skin. Injection tests of food extracts in these areas will be tried in the future.

CYTOLOGIC STUDIES.

Cytologic studies on food allergic patients by Bryan and Bryan[9] indicate that the presence of mast cells in nasal mucosal secretions may be significant. Oral secretions had not been investigated. Mast cells seem to play a part in the repair of injured tissues and may be associated with the mucopolysaccharides of the ground substance. Their association with heparin and histamine is well known, but the relationship of these substances to food allergy is obscure.

The cytotoxic test of Black, as performed by the Bryans,[10] indicates that leucocytes involved in the test may contain a factor which may be similar to the "transfer factor" described by Lawrence. The amount of antigen used can apparently influence the degree of reaction. It seems also influenced by whether or not the patient is having a reaction to the food in question. Blood, taken when the patient is apparently having an allergic response to an ingested food, was found to be non-reactive to the allergen; whereas blood taken the day before or the day after gave strong reactions. Since serum is also involved in the test, this may indicate that a high serum antibody titer may be present which neutralizes the antigen, thus making it unavailable for reaction with the leucocyte fixed antibody factors.

HYPERPLASTIC TISSUE CHANGES IN BORDERLINE ALLERGY.

In 1940 Fox and associates[11] described hyperplastic tissue changes in the nose and pharynx which they attributed to subclinical allergy and to which they applied the term borderline allergy. Careful studies were made of the formed elements in the mucosal discharge from the patients studied. The absence of eosinophilia was in striking contrast to that

found in patients with so-called frank allergy. The predominant cells found in these secretions were lymphocytes, plasma cells, histocytes, polymorphonuclear leukocytes and other wandering macrophages. Histologic studies of biopsy specimens taken from the noses of the subjects studied revealed the presence of a marked hyperplastic process. All connective tissue elements were increased, but classic edema of the tunica propria was missing, except to a slight degree. The cellular elements showed a preponderance of round cells. Histocytes, fibroblasts, plasma cells and neutrophilic leukocytes were present. The number of blood vessels were much increased and showed a high degree of periarteritis.

This description published by Fox 20 years ago is a classical example of the histologic picture seen in tissues involved in the delayed type of reaction. From personal contact with the chief author (Fox) at the time, we know that he did not suspect food as being the primary etiologic agent, nor was he aware of the true nature of the allergic type of reaction he had described. The article does mention food as a possible contributing antigen. Skin tests were found to be questionable and reliance upon trial diets and ingestion tests was recommended.

POSSIBLE SIGNIFICANCE OF LYMPHOID HYPERPLASIA.

Waksman[12] states that the delayed sensitization process takes place in lymph nodes which drain the site of inoculation. Characteristic delayed reaction is made up of perivenous accumulations of lymphocytes and histocytes. Sensitivity appears to be correlated with functional activity of circulating "sensitized" cells, possibly lymphocytes. It can be passively transferred by transfer of living lymphoid cells from sensitized donors to normal recipients. These findings suggest that sensitivity is mediated by circulating, sensitized mononuclear cells, lymphocytes and/or monocytes which accumulate where antigen is present, proliferate and give rise to secondary effects.

This concept of Waksman's lends support to our views, for lymphoid hyperplasia of the oral mucous membrane associated

with cervical lymphadenopathy is a constant finding in our patients with proven or suspected food allergy. Fox likewise was impressed by the extent of lymphoid tissue and lymphocytic cell involvement in patients with the allergic state which he described.

DISCUSSION.

We subscribe to the well documented and universally accepted concept that the ingestion of a specific food can elicit an immediate type of reaction in a food sensitive individual; however, not all of the clinical findings observed in these patients can be explained on this mechanism alone. We have attempted to show that the more obscure and less understood manifestations of food allergy could be an expression of a delayed type of hypersensitivity reaction. It has been suggested that the oral mucosa and possibly the mucosa of the esophagus and lower gastrointestinal tract may be involved in this process. Little has been done experimentally on man or animal to refute or support such a contention. Direct challenge of the oral mucosa by injection of extracts made from suspected foods might shed some light on this problem.

We realize that the views we have proposed are as controversial as the experimental and clinical evidence cited to support them. Yet the entire field of collagen diseases, which many assume to be of allergic origin, is no less clear. Speculation as to whether food sensitivity could play a role in the etiology of collagen diseases is beyond the scope of this paper. Such a concept remains an intriguing possibility.

CONCLUSION.

Clinical and experimental evidence has been presented to support the view that food allergy may elicit both the immediate and delayed type of reaction in the food sensitive individual. This concept was formulated in an attempt to clarify some of the obscure and little understood aspects of food sensitivity observed by the otolaryngologist.

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DISTANT METASTASES FROM PRIMARY MALIGNANCIES OF THE ENDOLARYNX.*

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R. A. Willis[1] in his book, "The Pathology of Tumors," in 1948 states that remote metastases in epidermoid carcinomas of the larynx are rare, but have been recorded in lung, liver and other organs. Later in a publication[2] "The Spread of Tumors in the Human Body," in 1952, he makes no mention of distant metastases of laryngeal tumors.

J. A. Maher[3] in the "Annals" of March 1960, reviews the subject of epithelial neoplasms of the larynx on a basis of necropsy material from 1923 to 1956. He cites 47 cases of carcinoma in which distant metastases were present in nine. The lesions involved the lung, liver, adrenal glands, bone and thymus; however, he makes no mention as to the exact site of the primary lesion.

Hamilton and Oppenheim[4] in an article "Carcinoma of the Extrinsic Larynx—Emphasizing Metastatic Disease" in the American Journal of Surgery in 1955, studied 61 cases of laryngeal malignancy. Six instances of distant metastases involved lung and liver, but only in one case did the primary site fall within our definition of endolaryngeal. The authors emphasize the fact that they believe a much larger number of cases with distant metastatic foci would be uncovered if more cases came to the autopsy table.

In 1948, Terricol and Bringer[5] reviewed the international literature from 1878 through 1947. They found reported only

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14 cases of distant metastases arising from carcinoma of the larynx. The exact primary site was unconfirmed; of these 14 metastatic cases only eight were histopathologically confirmed. The metastatic sites most frequently involved were lung and liver. Sawyer, *et al.*,[6] in 1954, reported a case of primary subglottic carcinoma. Eight months after a total laryngectomy, the patient was found to have metastases to the colon, and subsequently additional lesions were found in the lung, liver and small intestine.

Rubenfeld and Kaplan[7] in 1952 reported two cases; in one, the primary lesion was in the left ventricle. Subsequently the lymphatics of the left axilla became involved. No mention was made of other metastatic sites. In a second case, the primary lesion involved the false cords bilaterally. This case developed metastatic spread to the skin of the anterior chest wall. Again no mention is made of other definite metastatic sites.

J. W. Cavallaro[8] in 1960 reported a case of metastatic involvement of the myocardium and lungs, arising from a primary lesion located on the left true cord which extended into the left pyriform sinus.

On the basis of extensive review of the pertinent literature and various clinical inquiries and observations, the general opinion is that malignancies of the endolarynx exhibit a characteristic behavior of local extension and metastases to the cervical lymphatic chains. It is generally agreed by most that distant metastases are rare and usually occur preterminally.

The cases of carcinoma of the larynx at the Veteran's Administration Hospital at Los Angeles were reviewed for the years from 1950 through 1960. During that period there were 307 live discharges and 123 deaths, with 105 autopsies. In reviewing the autopsies, 23 cases of endolaryngeal carcinoma were studied in which eight cases were found where a primary lesion of the endolarynx developed distant metastases. These cases were autopsied at the Veteran's Administration Hospital in Los Angeles. Six of the eight cases were

initially examined and biopsied at the same hospital. Two were transferred from other Veteran's Administration Hospitals with complete records and biopsy slides.

In this series the range of ages at which the onset of symptoms occurred coincided with the usual distribution of tumors of the endolarynx. Age of onset of first symptoms: 50-54 years, two; 55-65 years, three; over 65 years, three. All eight cases presented initially complained of hoarseness as their first symptom. In some cases it was the only complaint. Other symptoms and findings included:

Hemoptysis	2.
Dysphagia	3.
Pain	2.
Weight loss	3.
Cervical adenopathy	1.

Five of the eight studied showed the presence of symptoms for a period of less than one year before the time of original diagnosis. In one instance symptoms had been present for more than one year before diagnosis was established, and in one case it was impossible to ascertain the time interval between onset of symptoms and establishment of diagnosis. None of these patients gave a history of alcoholic intake; four were heavy smokers and two were non-smokers.

In only one instance was another primary malignancy present. It was a lesion of the skin of the face and there was no evidence of metastatic behavior. None of these patients received any therapy for laryngeal carcinoma prior to their initial definitive diagnosis.

All eight cases at the initial examination showed involvement of one or both true vocal cords. In four cases, the neoplasm was confined to one or the other true cord. Three had extension from the cord into the ventricle. Three involved the false cord area and one had a subglottic extension. Three of the eight cases were unilateral; five were bilateral. Only one case had unilateral cervical nodes. In no case was there evidence of distant metastases at the time of the initial diagnosis.

The diagnoses were established on tissue taken by biopsy; in seven cases by the direct method; in one by the indirect method. Seven cases were diagnosed by the pathology department as differentiated squamous cell carcinoma; one as undifferentiated squamous cell carcinoma. Four of the cases were treated by total laryngectomy; four by irradiation in adequate amounts.

In two cases, recurrence or persistence of the original lesion was noted within six months—one case had had surgery; the other irradiation. The remaining six cases had recurrence within one year. Three cases of these manifested their initial recurrence in distant metastases, three in cervical nodes, and in only two was local recurrence evident. In the three cases of cervical metastases, all underwent radical neck surgery. One patient with local recurrence who had previously been treated by irradiation, was given additional palliative X-ray therapy.

Only four patients survived the first recurrence to demonstrate further evidence of malignant metastases. In three of these four cases, the second recurrence occurred within one year of the completion of therapy for the first recurrence. In the fourth case, the recurrence was in the second year. The sites of the recurrences were: one, local extension; one cervical metastases and two distant metastases. The case of cervical metastases was treated by radical neck resection. This case was the only one to survive his second recurrence; however, within one year he developed distant metastases for which he received irradiation, but soon expired.

At autopsy the persistence of the malignancy at or near the original site was present in five; cervical metastases were also present in five of the eight cases.

The composite list of the various sites of the metastatic foci other than direct extension, local recurrence or cervical metastases shows nine locations.

Lungs	8.
Liver	6.
Heart	3.

G.I. tract	2.
Bone	2.
Brain	1.
Adrenal	2.
Kidneys	2.
Skin	1.

COMMENT.

From this review of cases, we feel that distant metastases in primary endolaryngeal carcinomas are not so rare as usually maintained. This report could have included three or four times the number of cases had not the criteria been maintained of definitely establishing the primary site of the malignancy and taking only those cases carefully autopsied by one department at one hospital.

We were unable to uncover any statistical indication which would serve as a clinical guide to uncovering those lesions which would predispose to distant metastases. The clinical history and location of the primary lesion does not differ from the general behavior of carcinomas of the endolarynx. The method of therapy also fails to shed light on the question of whether distant metastases may be anticipated.

There are, however, a number of interesting factors in this study: the fact that all of these patients displayed pulmonary involvement. This may indicate that the distant metastases other than pulmonary arise from the secondary spread after the tumor has involved the lung, since it is well established that lesions in this area have a strong predisposition to blood-borne metastases; the frequent involvement of the liver and myocardial structures also is of great interest. Another factor which has been observed is the short survival of these patients as a group, considering the general behavior regarding survival in primary tumors of the endolarynx. In all instances it is felt that their reduced clinical course was due to the behavior of the distant metastases. We agree with Hamilton and Oppenheim[4] that a larger number of cases of this type would be uncovered if autopsy

percentages were increased in cases of primary carcinomas of the endolarynx.

SUMMARY.

The literature concerning distant metastatic spread of laryngeal malignancies is reviewed. Eight cases are evaluated where it has been satisfactorily established that the primary lesion arose from endolaryngeal structures. The metastatic sites of other than cervical nodes are enumerated.

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RHEUMATOID ARTHRITIS OF THE CRICO-ARYTENOID JOINT.*

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There is little to be found in the literature on the subject of crico-arytenoid arthritis. Until recently, only casual reference was made to this condition in some ear, nose and throat textbooks and journals.[1,2]

Photographs of crico-arytenoid joints manifesting arthritis due to rheumatoid or non-rheumatoid causes are rare, and this paper is designed in part to present such material along with authenticated case histories of rheumatoid arthritis with crico-arytenoid joint (laryngeal) manifestations.

Granting that laryngeal complications developing from rheumatoid arthritis are rare, one wonders why, when they do occur, they have not been diagnosed more frequently by physicians and laryngologists *during the patients' lifetime*, especially when such patients at autopsy exhibit extensive crico-arytenoid joint changes due to the generalized disease process.

One could try to explain this "diagnostic oversight" on the grounds that patients with rheumatoid arthritis have many obvious generalized symptoms and signs, such as distorted joints and numerous severe pains related to the arthritis in various parts of the body which may overshadow the inconstant, vague laryngeal symptoms produced by crico-arytenoid arthritis.

We have routinely examined the larynx and carefully ques-

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tioned 55 patients with rheumatoid arthritis (American Rheumatism Criteria 1959),[3] and we have found that rheumatoid arthritis of the crico-arytenoid joint causes few laryngeal symptoms. Seventeen patients manifested some symptoms referable to the larynx, but 38 presented without laryngeal complaints. On mirror examination, 18 patients revealed some suggestive evidence of crico-arytenoid arthritis, and half of these (nine) were patients who presented without laryngeal complaints.

Undoubtedly, the disease can cause major distortions and ankylosis in the crico-arytenoid joint which may restrict joint function and lead to cord fixation either unilaterally or bilaterally; *however, even such advanced changes may produce few laryngeal symptoms to direct the clinician to suspect arthritic complications in the larynx.*

It is generally recognized that fixation of one true vocal cord in the midline will not produce significant hoarseness nor stridor. This is also true for bilateral midline fixations if the patient is bedridden and not exerting himself.

Our investigations on the above series of 55 outpatients and on five other rheumatoid arthritics who came to autopsy have led us to the following conclusions:

1. Patients with rheumatoid arthritis may have intense microscopic involvement of the crico-arytenoid joint *without laryngeal symptoms.*

2. This occult complication of rheumatoid arthritis should be sought for *routinely* by the laryngologist and the clinician whether or not there be voice change, dyspnoea, stridor, dysphagia, or discomfort in the throat.

3. Laryngeal examination may be most unrevealing but should be undertaken to ascertain whether or not the crico-arytenoid joints are grossly involved in cases of rheumatoid arthritis, but only where indirect mirror examination is feasible and possible. This is for educational and clinical completeness, if, at times, for nothing else.

Fig. 1a. Note pannus* formation and irregular destruction of the cricoid articular surface, with suppuration and purulent exudate within the joint cavity.

SYMPTOMS.

Most authors describe symptoms which relate to this condition and these seem logical to expect; however, in our series, few patients gave a history of laryngeal symptoms, nor did they show clear, consistent, significant, gross objective changes locally by mirror examination to suggest crico-arytenoid arthritis (*i.e.*, ankylosis, deformities, joint swelling, or cord fixations).

*"Pannus" is the extension of a chronic synovitis from the joint margins along the articular surfaces which are thereby destroyed and replaced by chronic inflammatory fibrous tissue. (H.R.)

Fig. 1b. Note the chronic synovitis and marginal destruction and replacement of the articular surfaces by pannus. There is fibrinoid degeneration with debris of a non-suppurative nature in the joint cavity.

Below are some of the symptoms which one expects to be present, but more often than not are absent, as verified in our above-mentioned series.

1. Persistent sore throat.

2. Feeling of "something sticking in the throat" after the act of swallowing has been completed.

3. Dysphagia. This may occur when there is thickening, swelling, and distortion of the arytenoid and crico-arytenoid joint.

4. Hoarseness. If one or both vocal cords are free of disease and become fixed in the midline position, there will usually be little, if any, voice change. If one or both cords become fixed in the lateral position, then one usually finds hoarseness. From our clinical results it would appear that voice change is not a common finding in crico-arytenoid arthritis.

Fig. 2. Note the erosion of the arytenoid articular surface, synovitis, pannus, and fibrinoid debris in the joint space.

In only two of the five cases which came to autopsy and revealed crico-arytenoid arthritis, did hoarseness or any other symptom appear as a clinical laryngeal complaint, and yet all five cases demonstrated microscopic evidence of rheumatoid arthritis of the crico-arytenoid joint (see Figs. 1a, 1b, 2, 3, 4, 5).

5. Stridor. In relatively early rheumatoid arthritis cases when the patient is still ambulatory and able to work, we might expect that, if there is a midline fixation of both vocal cords, stridor might develop on physical exertion. Examina-

tion of the larynx would demonstrate the midline bilateral true cord fixation along with any other changes that may involve the crico-arytenoid joint.

In the more advanced cases of rheumatoid arthritis, and this sounds paradoxical, when the patient is bedridden it is a different story. Now we find that both vocal cords may be fixed in the midline, but the patient is *not able* to exert and

Fig. 3. Note the marked fibrosis and relatively little active inflammatory reaction in a partially ankylosed joint, with disorganization of the articular surfaces, and interposed fibrous tissue.

does not have enough activity to induce stridor. At rest, the patient may, therefore, have a clear voice, and breathe without stridor, but still have an extensive occult laryngeal involvement due to crico-arytenoid arthritis; therefore, on the clinical grounds of stridor and voice change alone, one cannot determine whether or not a patient has developed arthritis of the crico-arytenoid joints with cord fixation.

Darke, *et al.*,[4] noted that most of his cases of stridor described as suffering with midline cord fixations clinically

due to arthritis were proven at autopsy to be cases of bilateral recurrent nerve abductor paresis. In one case, cancer of the larynx was the cause of the stridor and not arthritis of the crico-arytenoid joint. We emphasize this because recurrent laryngeal nerve damage producing cord fixations must be considered and excluded in differential diagnosis of arthritis of the crico-arytenoid joint, and sometimes as Darke has proven, this may be clinically difficult.

Fig. 4. Note the thickened synovium with marginal pannus which tends to undermine the articular cartilage, and the fibrinoid debris in the joint space.

SIGNS.

1. There are reports in the literature which state that in rheumatoid arthritis, laryngeal mirror examination may reveal redness and swelling of the mucosa over the crico-arytenoid joint. This can be unilateral or bilateral, and the swelling can be extensive enough to interfere with the act of swallowing, but in our series examined by laryngeal mirror this finding was noted only rarely.

2. The crico-arytenoid articulation in some cases of rheumatoid arthritis may become disorganized and the joint ankylosed in a deformed position.

3. Fixation of one or both true vocal cords may occur in the midline position.

DIFFERENTIAL DIAGNOSIS.

The diagnosis of rheumatoid arthritis of the crico-arytenoid joint should be based on a complete history, physical exam-

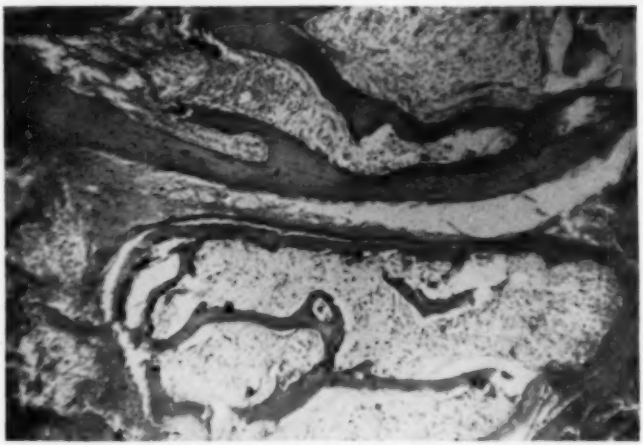

Fig. 5. The cartilages are extensively ossified and there is associated chronic inflammatory reaction with exudative and productive changes in bony and ligamentous tissues, commensurate with a severe arthritis. The cellular exudate of large mononuclear cells and polymorphs is noteworthy. There is no suppuration. Note also the extensive fibrosis of the joint space.

ination, blood tests, and differential diagnosis in which careful attention is paid to the mirror examination of the vocal cords and crico-arytenoid articulations.

Central and peripheral changes in the vagus and recurrent laryngeal nerves which produce true cord fixations should be excluded. In this respect one must consider cerebral, neck, oesophageal, mediastinal, and cardio-pulmonary lesions which

can involve the recurrent nerves and produce true vocal cord fixations.

The differential diagnosis should consider non-rheumatoid conditions that may occasionally occur and produce swelling and/or ankylosis of the crico-arytenoid joints. One example is the edema of the arytenoid area which often develops

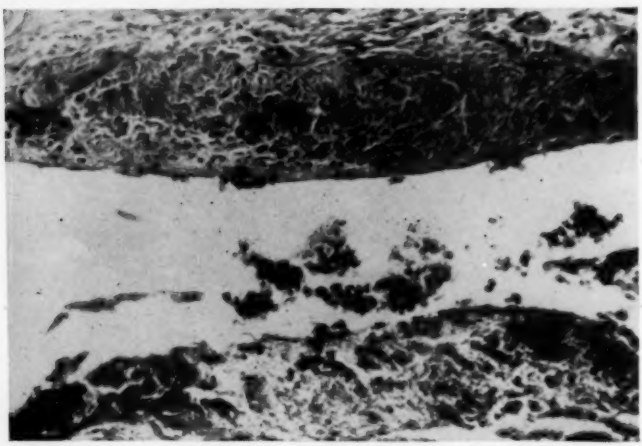

Fig. 6. Note the thickening of the synovium with little or no inflammatory cell infiltration but with proliferation of the synovial cells, much fibrinoid deposition (or necrosis) within and on the surface of the synovium, and much necrotic debris in the joint cavity.

after irradiation treatment for malignancy of the larynx. Crico-arytenoid ankylosis from disuse is another.

Laryngeal injuries during laryngoscopy and bronchoscopy, or resulting from foreign bodies and their removal, or injuries due to prolonged or traumatic Levine tube feeding, or to endotracheal intubation for anesthesia, are all situations that may and can lead to crico-arytenoid oedema, arthritis, and ankylosis of a non-rheumatoid type. Occasionally suppurative crico-arytenoid arthritis may result from upper and/or lower respiratory tract infection. These conditions,

therefore, must be considered in the differential diagnosis of rheumatoid arthritis of the crico-arytenoid joint.

Recently, in a patient who died from a collagen disease (scleroderma), a routine post-mortem examination of the crico-arytenoid joints revealed an unsuspected non-suppurative arthritis (see Fig. 6). This suggests that arthritis of a non-suppurative type can be found in conditions other than rheumatoid arthritis.

PATHOLOGY.

In rheumatoid arthritis there occurs a characteristic synovitis with both proliferative and exudative features. The synovium is thickened by fibroblastic proliferation and infiltration of lymphocytes and larger mononuclear cells or histocytes, the former frequently in focal aggregations as well as less dense diffuse dispersion, and the latter, frequently elongate, and arranged in palisade fashion with long axes parallel to one another and at right angles to the plane of lining surface. There may be fibrinoid alteration or necrosis of superficial portions, and fibrinoid material in the joint cavity.

The chronic inflammatory fibrosis of the synovium encroaches across the articular surfaces, destroying and replacing the articulating cartilages; this inflammatory membrane is known as pannus. Chronic inflammatory reaction may also progress inward along the deep aspects of the cartilages. Pannus may obliterate the joint cavity, and with increasing fibrous density, produce fibrous ankylosis. This may in turn be replaced by bone, the end result being unyielding bony ankylosis and deformity. (H.R.) (See Figs. 1b, 2, 3, 4, 5.)

For comparative purposes, Fig. 7 reveals the characteristics of a normal crico-arytenoid joint.

TREATMENT.

In addition to the general measures employed in the treatment of rheumatoid arthritis, the laryngologist can provide tracheotomy if the patient manifests stridor due to bilateral

midline fixation of the vocal cords. The tracheotomy would also facilitate aspiration of secretions from the lower respiratory tract to prevent recurrent pneumonia.

The laryngeal surgeon can also perform arytenoidectomy and vocal cord lateralization procedures where indicated in order to close the tracheotomy, and at the same time ensure a safe airway and preserve the patient's voice.[5]

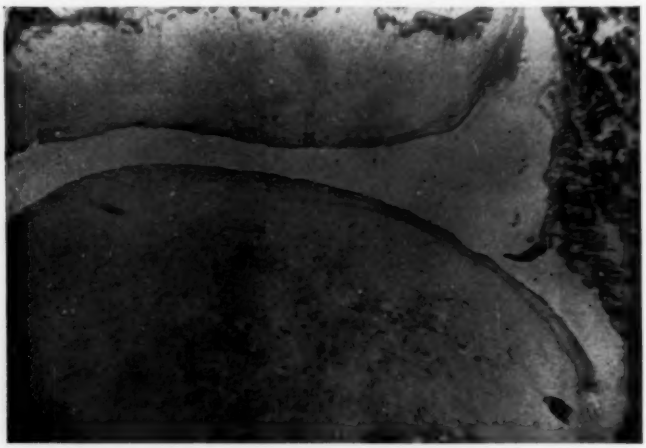

Fig. 7. Note the convex margin of the arytenoid and the correspondingly concave cricoid cartilage. There is a thin and uniform synovium with underlying dense fibrous tissue. The joint cavity is empty; the articular surfaces are slightly separated here due to artefact. The articular surfaces are smooth and regular. The synovium is present at the edges and disappears over the articular cartilage. Note the fornix of the joint lined by synovium.

Some arthritic patients, because of the deformity of their fingers, may have difficulty caring for their tracheotomy lumen in regard to speaking, and to wiping away of secretions at the orifice of the tracheotomy. In such cases, vocal cord lateralization operations are of great benefit.

These patients are not particularly interested in the cosmetic features of the tracheotomy opening in the neck, since they suffer with generalized joint changes that are more deforming, and much worse from an aesthetic viewpoint than

the tracheotomy orifice. It is for the above-mentioned and much more practical reason that they welcome a vocal cord lateralization procedure, with closure of the tracheotomy orifice.

CASE REPORTS.

At the Montreal General Hospital in the past two years, five of 11 consecutive cases of rheumatoid arthritis who have been autopsied demonstrated evidence of rheumatoid involvement of the crico-arytenoid joints. These five cases are presented below. Only two of these cases gave a history of hoarseness—the others were free of laryngeal symptoms.

Case 1. (088835) Mrs. N.D. A 74-year-old rheumatoid arthritis patient was admitted to the Montreal General Hospital June 2, 1958, and died June 8, 1958. On examination, it was noted that she was a "definite" case of rheumatoid arthritis.[5]

A small sinus discharging purulent material was observed on the postero-lateral aspect of the right ankle. (Staph. pyogenes was cultured from this.)

She had never been hoarse nor did she complain of stridor or dysphagia.

Pathology.

The autopsy revealed widespread rheumatoid arthritis, with a superimposed suppurative process involving the knee, the right heel, *and the right crico-arytenoid joint.* Staph. pyogenes was cultured from the synovial exudate of the right crico-arytenoid joint. (Fig. 1a is a section of the right crico-arytenoid joint, and Fig. 1b of the left crico-arytenoid joint.)

COMMENT.

1. There were no clinical symptoms suggesting crico-arytenoid involvement in this case in spite of the extensive arthritis later found in both joints at post-mortem examination.

2. Suppurative arthritis complicating rheumatoid arthritis has been described by Kellgren, J. H., *et al.*,[6] but never has such a complication been described involving the crico-arytenoid joint.

Case 2. (090714) Miss H.S. A 57-year-old white female was admitted to the Montreal General Hospital June 30, 1958, and died the same day.

In the Spring of 1958 various digits of the hands and feet became gangrenous. It was believed that she was suffering from a vasculitis as described by Irby, *et al.*,[7] and Bywaters.[8]

On admission, most of the joints were painful, swollen, and almost

immobile. A sensory and motor neuropathy involving the hands and feet was present. Most of the fingers were gangrenous. There were no juxta-articular nodules.

In spite of a careful observation regarding laryngeal symptoms, it was noteworthy that at no time was the patient hoarse, nor did she have any trouble swallowing.

Pathology.

The pathological examination revealed a widespread vasculitis involving many organs. Both crico-arytenoid joints were involved by rheumatoid arthritic changes with no evidence of ankylosis (see Fig. 2).

COMMENT.

Her case history is noteworthy in that there were no clinical symptoms to suggest rheumatoid involvement of the crico-arytenoid joint even though these had been carefully looked for before her demise.

Case 3. (163543) Miss V.O'H. A 40-year-old single white female was admitted to the Montreal General Hospital February 2, 1959, for surgical correction of joint deformities which had resulted from rheumatoid arthritis.

She had developed rheumatoid arthritis 15 years previously, with involvement of almost all of her joints. She had been under long-term treatment with adrenal cortical hormones, and at the time of her admission she was on Dexamethosone.

She had been hoarse for several years but there was no stridor nor difficulty with swallowing or breathing. In recent months she had become bedridden due to her deformities.

On examination the hoarse voice was quite prominent. The patient fitted into the category of "definite" rheumatoid arthritis.[3]

Because of the hoarseness, it was assumed that she must have developed rheumatoid arthritis of the crico-arytenoid joints with fixation of one or both vocal cords. The lack of stridor was noteworthy, suggesting cord fixation in the cadaveric or lateral position.

One of us (A.G.) tried to visualize the vocal cords, but due to the rigidity of her neck and jaws caused by the disease, indirect mirror laryngoscopy was impossible and direct laryngoscopy was not attempted.

The patient died shortly after admission.

Pathology.

The autopsy examination of her larynx revealed bilateral crico-arytenoid arthritis. Fig. 3 is a section of one crico-arytenoid joint.

COMMENT.

1. The above case illustrates the typical findings of ankylosis in crico-arytenoid arthritis from a histopathological standpoint.

2. It also emphasizes that due to arthritic deformities, it

may be impossible to examine a larynx in order to determine whether or not cord fixation and/or crico-arytenoid joint changes have resulted.

3. This patient was one of the five post-mortem rheumatoid arthritis cases here presented, who in life manifested laryngeal symptoms which suggested crico-arytenoid arthritis. Only one other of these five cases displayed laryngeal symptoms.

Case 4. (050814) Miss E.E. A 77-year-old white lady with "definite" rheumatoid arthritis[3] was followed at the Montreal General Hospital until her death from intestinal obstruction in July 1959.

She was quite depressed and had a mild degree of Parkinsonism with muscle rigidity and a monotonous voice. There was also some bilateral conductive deafness due to otosclerosis—or possibly to arthritis of the conductive ossicular chain.

In the last year of her life, close attention was paid to her voice. It never changed from the Parkinsonian monotonous tone. She complained of alternating periods of excessive salivation followed by dryness of the mouth. There was no dysphagia, no stridor, and no hoarseness.

Fig. 4 reveals typical histopathological findings of crico-arytenoid arthritis.

Case 5. (038217) T.S. Age 36 years. This white male patient was in good health until the age of 22, when he developed ankylosing spondylitis, with arthritis, presenting first in the peripheral joints but progressing to involve the spine and nearly all joints, so that by 1955 he was completely bedridden. In 1950 a bilateral iritis occurred, with resultant loss of vision in the left eye.

X-rays demonstrated the typical changes of rheumatoid spondylitis in the sacro-iliac joints and vertebral column. Early in 1958 gross oedema appeared in both legs. There was also marked proteinuria, lowered serum albumin, and impaired urinary concentrating power. Secondary amyloidosis was suspected, and the patient was discharged on Prednisone 5 mgms. b.i.d. He gradually became stuporous and died two months later (Nov. 7, 1958).

At autopsy, the clinical diagnosis of rheumatoid spondylitis with peripheral joint involvement and with secondary amyloidosis of kidneys and other organs was substantiated.

From the laryngeal standpoint, it is interesting that from March 1958 to July 1958 he complained of intermittent bouts of hoarseness.

His larynx was not investigated before his demise, the symptom hoarseness being so minor in the face of his many more serious complaints.

Fig. 5 is a view of one crico-arytenoid joint.

COMMENT.

Vague intermittent hoarseness was present but overlooked clinically. This symptom certainly was slight considering the extensive changes later found on histopathological study of the crico-arytenoid joints.

Fig. 6 is a section of a crico-arytenoid joint demonstrating non-suppurative arthritic changes in a patient who had suffered with a collagen disease (scleroderma), *without any laryngeal symptoms.*

This is inserted to demonstrate that there can be non-suppurative crico-arytenoid arthritis due to causes other than rheumatoid arthritis.

Fig. 7 is a section of a normal joint presented to serve the reader with a histopathological base-line for comparative purposes.

CONCLUSIONS.

1. Some suggestions are offered to account for the occult nature of crico-arytenoid arthritis of the larynx.

2. Five case histories of rheumatoid arthritis with crico-arytenoid changes are added to the literature.

3. A case of scleroderma (collagen disease) with crico-arytenoid arthritis is recorded.

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APPLICATION OF FLUOTHANE ANESTHESIA TO OTOLARYNGOLOGICAL SURGERY.*†

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The anesthetic agent fluothane, or halothane, was introduced by Raventos in England in 1956.[8] In that year a few institutions in the United States began to utilize it and observe its clinical effectiveness. One of these investigating institutions was the University of Vermont, together with its associated hospitals, the agent having been utilized extensively here since that time. Over 15,000 anesthetizations have been given, involving operations in all surgical fields. As we worked with the agent, we found that it had characteristics which rendered it especially advantageous in the operations of otolaryngology, as will be brought out subsequently.

In many institutions this agent has been given as a supplement to nitrous oxide. We feel that this technique is not true fluothane anesthesia and fails to reveal the advantages of the agent. The nitrous oxide, being a very weak anesthetic, has little if anything to offer in the presence of this most powerful agent, and may predispose to anoxia. It occupies gas volume which can be much more advantageously assigned to pure oxygen. We use oxygen alone as a diluent, and feel that this is of great value in preventing serious complications. If respiration is interrupted while the pulmonary "dead space" is occupied by oxygen rather than air or another partly inert

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mixture, the state of apnea may be continued considerably longer without detrimental effect on the patient.

It is important that the method of administration be of a type that provides constant information as to the concentration of fluothane; *i.e.*, accurate vaporizing equipment. In our early usage of the agent we employed semi-closed or non-rebreathing methods and the only complications in our experience occurred during these early days. In those days we had three incidents of cardiac arrest in the entire experience of all surgical fields. In two of these patients there was prompt recovery on cardiac massage and they underwent the intended surgery successfully at later dates. The third was a case of ruptured aortic aneurysm being repaired under hypothermia. Asystole developed when the aneurysm was opened after four hours of occlusion, and it was felt that fluothane had nothing to do with this event. There have been no serious complications since the adoption of a closed, controlled method of administration.

Induction with the agent is very smooth and rapid. In children a playful approach is possible with a "space helmet," or the delivery tube of the gas machine may simply be held near the face while the anesthesiologist talks with the patient.

The preanesthetic routine which we use is a mixture of alphaprodine (Nisentil), 40 mg. in the adult or 24 mg. per 50 pounds in a child; scopolamine, 0.4 mg. in the adult, 0.5 mg. per 50 pounds in the child; and levallorphan (Lorfan) 0.8 mg. in the adult and 0.15 mg. per 50 pounds in the child. These are given intramuscularly 45 minutes preoperatively.

One cannot be careless with this agent. With proper equipment in the hands of an alert, trained anesthesiologist, however, it is entirely safe and in many ways quite superior in otolaryngologic surgery. The application to specific types of cases will be given below. The operations included in this report are listed (see Table I) as are the specific advantages of the agent (see Table II).

TONSILLECTOMY AND ADENOIDECTOMY.

Tonsil and adenoid surgery is somewhat unique in its requirements of an anesthetic agent. The ideal agent should be one with ease of administration, minimal odor and rapid effectiveness, especially since most of our patients are children, tending to have a certain degree of apprehension, some

TABLE I.

Operations Done with Fluothane.

T. and A.	1672
Endoscopic Procedures	217
Laryngectomy (mostly with neck)	29
Neck Dissections	24
Mastoid and Ear Operations	191
Maxillofacial and Major Sinus Operations (including oral cancer, etc.)	129
Miscellaneous Major Operations	61
Miscellaneous Minor Operations	114

TABLE II.

Special Advantages in E.N.T. Surgery.

Rapid Recovery <	Rapid, non-irritating induction.
	Cough—5 min.
	Talk—15 min.
	Suppression of throat reflexes.
	Lack of salivary and bronchial secretions.
	Easy control of anesthesia level.
	Administration with O_2.
	Lack of vomiting in induction and recovery.
	Non-explosive.

having been known to experience psychic trauma from this type of surgery. Fluothane meets these requirements and, in addition, offers advantages of considerable magnitude to the surgeon. In proper concentration depression of cough reflex, inhibition of movements of the paryngeal muscles and absence of excessive mucous and salivary secretion are readily obtained. The plane of anesthesia can be increased or decreased rapidly; in fact, within a matter of seconds, by regulating the percentage of concentration of the agent; furthermore, when fluothane is stopped there is a rapid recovery of protective reflexes, thus tending to prevent postoperative complications.

Our records at the Mary Fletcher Hospital, since the Fall of 1956, indicate that a total of 778 tonsillectomy and adenoidectomy cases received fluothane anesthesia. Of this number 14 patients received a fluothane-ether-nitrous oxide-oxygen mixture; 13 received fluothane-ether-oxygen, and 47 received fluothane-nitrous oxide-oxygen. These combinations were given in the early months of its use but it was subsequently found that fluothane and oxygen was the ideal mixture, and this was given to the remaining 704 patients. There were no complications in this series.

Another series of cases operated upon at the DeGoesbriand Memorial Hospital totaled 869. Of this number 688 patients had a standard tonsillectomy-adenoidectomy, 120 had myringotomy in addition, 19 had tonsillectomy alone, and 36 had an adenoidectomy alone. In addition, there was one case for excision biopsy of a small tumor of the tonsil fossa and five cases for control of postoperative bleeding. There was only one complication in this group, a case of cardiac arrest mentioned above in a patient who received fluothane in combination with other anesthetic agents. This patient recovered and underwent the operation at a later date under fluothane.

There have been some instances of delayed postoperative vomiting, this occurring four to six hours after surgery. Since fluothane is eliminated from the system very rapidly, we suspect that something else is responsible for the vomiting. The fact that the patients react promptly and are taking fluids much earlier than cases which have had ether may be an explanation.

It is the impression of all of our surgeons doing tonsillectomy and adenoidectomy, and of the nurses in charge of postoperative care, that fluothane is superior to all previously used general anesthetic agents in this type of case.

Endoscopy:

The majority of peroral endoscopies in our hospitals are done under topical anesthesia. There are instances, however, where general anesthesia is preferred, and these cases present special problems. Relaxation of the jaw, the pharynx and

the larynx is necessary. Bronchospasm and laryngospasm are most interfering to the operator and dangerous to the patient; yet, the anesthesiologist is asked to control this situation while he is robbed of the closed system usually afforded him by the endotracheal technique.

Various solutions have been offered, for example:

1. Ether, the patient being maintained in the second or third plane of the third stage. This is unsatisfactory, because if the patient becomes too light the process of getting him back down to a relaxed plane is slow; furthermore, the view through a bronchoscope filled with ether fumes is wavy and blurred.

2. Topical, plus pentothal, plus a muscle relaxant. This is an apneic technique and carries all the underlying complications and hazards of the muscle relaxant.

3. The same, plus a cuirass-type respirator, with the difficulties introduced by complex equipment.

In our experience, fluothane has been most satisfactory in endoscopy. It has proven especially good for bronchoscopy in children.

The Advantages Are:

1. Rapidity and ease of induction in a child with compromised ventilation, as by cough and bronchospasm.

2. Full relaxation of the jaw, pharynx and larynx without resort to relaxants.

3. Full oxygenation while administering the anesthetic through the sidearm of the bronchoscope.

4. Quick restoration of relaxing level if the patient becomes light.

The Technique Is as Follows:

The child is induced to a relaxed level. A little cocaine is sprayed into the larynx and the bronchoscope is passed. The fluothane, 2 per cent in pure oxygen, is attached to the side-

arm of the bronchoscope. The endoscopist's thumb is placed over the mouth of the bronchoscope during inspiration and removed during expiration, until satisfactory relaxation and quiet breathing are obtained. If at any time during the procedure the patient shows signs of becoming light, the operator resumes the thumb-to-bronchoscope maneuver, usually for just a minute or two, until satisfactory relaxation is achieved. Within a few minutes after the removal of the bronchoscope the child is awake and coughing freely. There is no haste. The bug-bear of apnea is removed. The only precaution is that the operator himself must avoid inhaling too much of the fluothane gas.

Esophagoscopy is no problem, being done by routine endotracheal anesthesia. The specific advantages of fluothane are:

1. Relaxation of the jaw, pharynx and crichopharyngeus, while the general anesthesia is not very deep.

2. Minimal regurgitation movements of the esophagus.

3. Prompt recovery of the reflexes at the close of the anesthesia.

In laryngoscopic procedures we usually prefer topical anesthesia alone, with heavy pre-medication, but in those for whom general anesthesia is chosen, special problems arise. In many cases the laryngeal disease itself tends to limit the airway by occupying breathing space or by increasing laryngeal spasm because of pain, inability to absorb topical anesthesia, or increase of mucus in the area. In the induction, fluothane is ideal because it affords rapidity, minimal irritation of the mucosa and high oxygen supply; however, maintenance of anesthesia with fluothane with a wide open larynx is most difficult. A technique we formerly used was to establish a deep plane of anesthesia with ether, remove the mask and then do an endolaryngeal procedure while the patient gradually recovered to the level of moving the vocal cords. This is not possible with fluothane. A quick biopsy can be taken, but detailed studies or careful stripping of the vocal cords cannot be done. In cases requiring time we use a cuffed

endotracheal tube.* This is a small, silk-woven tube with a large balloon. The anterior-commissure type of laryngoscope fits comfortably into the glottis anteriorly to this tube, and any desired procedure may be done on the anterior two-thirds of the vocal cords. Inspection and biopsy can be done in any part of the larynx.

Laryngectomy:

In 1959, Boyan and Howland[4] published a paper on the management of anesthesia in laryngectomy, pointing out special dangers and problems in this operation. They spoke of apnea after tracheostomy, interference with the airway by the disease itself, the necessity for sharing the area with the surgeon, and the tendency of blood, secretions and tumor to enter the trachea. Their special danger times were the initial intubation and the transferring of the anesthesia from the oral approach to the transected trachea. Our approach has been to avoid these danger points by doing the operation up to the time of transection of the trachea under deep cervical block anesthesia, supplemented by local infiltration of xylocaine. When the trachea is transected, fluothane anesthesia is introduced by a cuffed tube inserted into the tracheal stoma. Any patient who has enough initial obstruction to make the operation under cervical block anesthesia inconvenient, is usually already tracheotomized prior to the main operation, and the fluothane can be delivered directly through the tracheal stoma; however, we have used initial tracheal intubation in a few patients for special reasons. Here fluothane, with its pure oxygen vehicle and its lack of irritation, laryngospasm and secretions, has proven most satisfactory.

The introduction of fluothane at the time of the tracheal transection has given us no difficulty. Again, it is the rapid, quiet induction, with minimal mucosal irritation, that makes this possible.

Obstructed Cases:

Fluothane is ideally suited to the special situation presented

*We note that Priest and Weslowski have also recently described this technique.[13]

by the need for general anesthesia in a case of respiratory obstruction; for example, acute laryngotracheobronchitis, retropharyngeal abscess, benign tumors of the larynx or pharynx, and, as recommended by some recent authors, the tonsillectomy in acute peritonsillar abscess.

The technique we formerly used in laryngotracheobronchitis was to do laryngoscopy without anesthesia, to pass a bronchoscope through the narrow glottis, and then to do the tracheostomy over the bronchoscope under locally injected anesthesia. In a large, restless, frightened child, one seriously questions whether to force the bronchoscopic technique or to go ahead with the struggle of a tracheostomy under local anesthesia on a stridorous, moving target, with the added danger that the physical struggling of such a child increases the oxygen demand. Once, in such a situation, our anesthesiologists proposed fluothane, since it could be given with a flow of pure oxygen without irritation and spasm, without premedication, and without vigorous restraint of the child. It was most pleasing to watch the patient breathing the oxygen without a fight, gently going off to sleep. We have done six cases by this method. We stood by with the bronchoscope, but it was not needed. In all cases the trachea was intubated by the anesthesiologist. Once this free, fully controlled exchange was established, tracheostomy could be done without haste or patient restraint.

Similarly easy induction with adequate oxygen flow has been done in cases of nasal or nasopharyngeal obstruction as well. A good example is the case of a four-month-old infant from whom we removed a large intranasal glioma. The key to these obstructed cases is, of course, that the patient situation during induction is about the same as during the normal inhalation of oxygen. There is simply added a slight flow of non-irritating anesthesia which slips him over into unconsciousness.

Very Short Cases:

Fluothane in oxygen is the ideal answer for such quick procedures as myringotomies, elevation of nasal fractures,

removal of ear polyps, painful ear dressings, and the like. Smooth, rapid induction, smooth rapid recovery, without persistent sedation or "hangover," yet with anesthesia deep enough to do the procedure without resistance movements by the patient, are the factors which make it ideal.

The following cases illustrate this situation:

A boy, age eight, was struck by a batted baseball, fracturing his nose. At four p.m. fluothane anesthesia was administered and the nasal fracture elevated. At six p.m. he was in the ball park watching the game, having eaten a hamburger on the way.

Another boy, age 13, who had been brought in for bilateral myringotomy, was one of the most unco-operative children we have ever encountered. When we finally "sold" him the idea of giving himself the gas, he held the mask, only to complain that the gas was not on. He could not smell anything. He was reassured that it would come soon and that if he did not like the smell he could remove the mask. While still "awaiting the gas," he fell into unconsciousness. One hour after this procedure one of us was in the hospital coffee shop when this child came gaily in and sat down and ordered a chocolate milk. We asked whether he was sure he could keep it down. A lady with him promptly assured us that he could, as he had already eaten a hamburger 15 minutes before. This originally terrified boy went home with a happy memory of the whole event.

Long Procedures:

Head and neck cancer work and reconstructive middle ear surgery are calling for longer and longer operations. Sustained general anesthesia is dangerous, but the danger diminishes according to the lightness of the level at which the patient is carried. Fluothane has proven most satisfactory in this regard. In abdominal surgery, 2 per cent fluothane is needed to maintain adequate muscle relaxation, but in work about the head and neck, the average maintenance concentration is only about 0.8 per cent. In one of our cases, where radium needles were introduced into the base of the tongue, and it was desired that the patient remain absolutely still until these were removed, the patient was carried under a very light fluothane anesthesia for 14 hours. The concentration of the gas was just barely sufficient to keep the patient below consciousness, but the quietness of the pharyngeal reflexes at this light level were such that the radium positions were not subjected to any hazard.

In connection with cancer surgery, it should also be men-

tioned that the electrocautery can be used with no risk in the presence of this gas.

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PSEUDOSARCOMA ASSOCIATED WITH SQUAMOUS-CELL CARCINOMA OF THE LARYNX, REPORT OF A CASE.

JOHN W. GRIGG, M.D.,
NIKOLAI RACHMANINOFF, M.D.,
and
JAMES MILTON ROBB, M.D.,

Detroit, Michigan.

Carcinosarcoma of the larynx has been infrequently reported in the literature. In 1957, Lane[1] introduced a new term for this entity, "pseudosarcoma associated with carcinoma." The sarcomatous stroma is regarded as a benign secondary reactive phenomenon, or response to the carcinoma. Pseudosarcoma is used to indicate, "a presumably non-neoplastic connective tissue mass with a histological appearance that is at once bizarre, 'alarming,' and deceivingly suggestive of some fully malignant form of true sarcoma capable of metastasis." Lane reported ten cases, four of which involved the larynx. He noted that the pseudosarcomatous portion lacked ability to metastasize. Significant features were not apparent in the patients' histories. Two had received previous irradiation, one 16 years previously for "leukokeratosis" of the tongue; the other, 30 years previously for tuberculous cervical adenitis. The prognosis for laryngeal cases was the same as for carcinoma of the larynx without sarcomatous change.

Although Lane is the first in the American literature to use "pseudosarcoma" with carcinoma, Dal Pozzo, cited by Saphir and Vass,[2] in 1931, in the Italian literature, used the term pseudosarcomatous carcinoma, or pseudocarcinosarcoma to describe carcinoma with sarcoma-like appearance.

Baker[3] in 1959 reported three cases of pseudosarcoma with carcinoma of the larynx. One was a follow-up of a patient

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previously reported by Lane. Frank and Lev[4] in 1940 studied 62 cases of carcinoma of the larynx and found 30 to have morphological variations of the squamous cells simulating sarcomatous appearance. They analyzed five cases of carcinosarcoma of the larynx from the world literature, and concluded these tumors were comparable to some of their carcinomas, but did not represent true carcinosarcomas. They

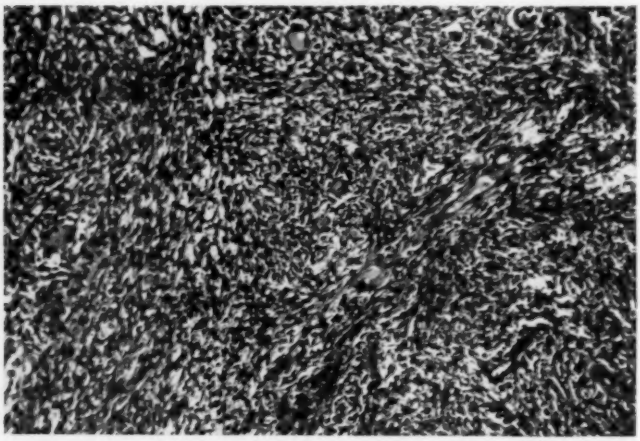


Fig. 1. Pseudosarcoma of larynx. "Sarcomatous" appearance of the stroma from the central portion of the tumor. Note spindle-shaped character of the cells. (H&E x 140.)

believed such tumors showed only morphological variations of the squamous cell element accompanied by an increased amount of connective tissue. In some, repeated examinations were necessary to identify the fundamental cell as epithelial in nature. Only one of the patients with sarcomatous change had received prior irradiation, while five of the non-sarcomatous group had received previous irradiation. A history of prolonged hoarseness was present in the group displaying sarcomatous change.

Saphir and Vass[2] in 1938 reviewed 153 cases of carcinosarcoma from the world literature. Six involved the larynx. They

also believed the sarcomatous appearance of these tumors was either due to morphological variations of the squamous-cell carcinomas, particularly of the poorly differentiated variety, or due to chronic irritation and inflammatory reaction of the stroma which was considered by some authors as part of the tumor. Only three or four of the reviewed neoplasms, none of which involved the larynx, could be designated true carcinosarcoma with metastasizing ability of the sarcomatous element.

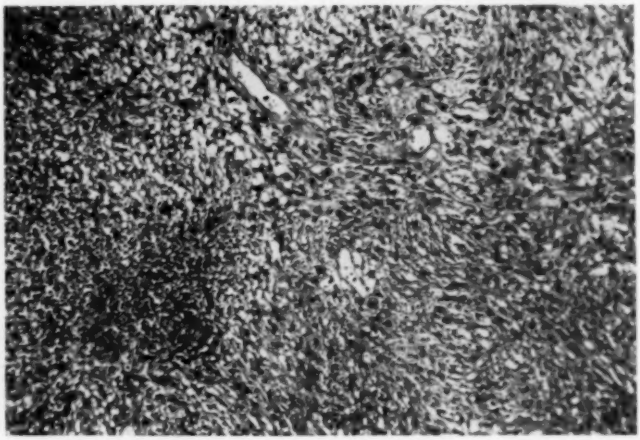


Fig. 2. Pseudosarcoma of larynx. "Sarcomatous" appearance of the stroma near the ulcerated surface. Note edema, vascularity of the lesion and inflammatory cells. (H&E x 140.)

All of the reported instances of carcinosarcoma or pseudosarcoma with carcinoma of the larynx occurred in persons over age 40, and most occurred over age 50. Carcinoma may be inconspicuous in the abundant, and sometimes predominantly sarcomatous-appearing stroma, and is readily missed if the examiner is not aware of this entity. The sarcomatous stroma, although appearing deceptively like a sarcoma, on close inspection reveals its benign nature. In the larynx, the lesion should be treated as a carcinoma and the sarcomatous changes disregarded.

Our case presents several interesting features. The lesion was seen by indirect laryngoscopy four years previously. The patient gave a prolonged history of hoarseness and had smoked three packages of cigarettes daily for years.

CASE REPORT.

A 48-year-old Armenian man who was a clerk in an automobile factory was admitted to Harper Hospital August 1, 1960, complaining of intermittent hoarseness of five and one-half years' duration, with persistent

Fig. 3. Pseudosarcoma of larynx. Higher magnification of the "sarcomatous" stroma showing frequent mitotic figures. (H&E x 320.)

hoarseness for the past four months. He also complained of discomfort when swallowing, but had no actual difficulty in swallowing. The patient was otherwise asymptomatic. A history of smoking three packages of cigarettes daily for years was elicited. Four years previously he had consulted the attending otolaryngologist for intermittent hoarseness. Indirect laryngoscopy revealed whitish plaques on both vocal cords, especially on the right, which looked like leukoplakia. Direct laryngoscopy was advised, but he failed to keep the appointment for hospitalization, and was not seen again until the week prior to admission. General physical examination was normal. Indirect laryngoscopy revealed a smooth, grayish, pedunculated mass approximately two centimeters in diameter arising from the entire anterior surface of the left vocal cord which extended to the anterior commissure. A smaller, but similar lesion was present near the central portion of the right cord. Laboratory studies including a chest X-ray were within normal limits.

The tumor masses were removed under direct laryngoscopy. The patient's postoperative course was uneventful. Pathological diagnosis was superficial squamous-cell carcinoma of the larynx with underlying pseudosarcomatous changes.

The patient refused surgery and subsequently was treated with irradiation therapy. X-ray studies of the larynx prior to irradiation therapy revealed, "thickening of the true vocal cords, more prominent on the right with distortion of the laryngeal ventricle and thickening of the lower aryepiglottic fold on the right that is compatible with neoplastic infiltration; however, the possibility of edema from an inflammatory process, or following instrumentation would also have to be considered."

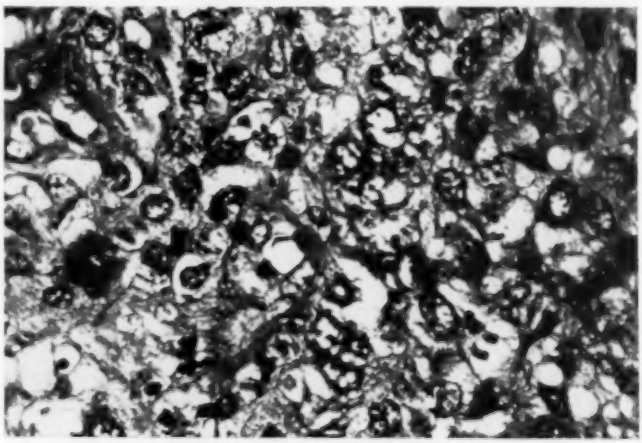


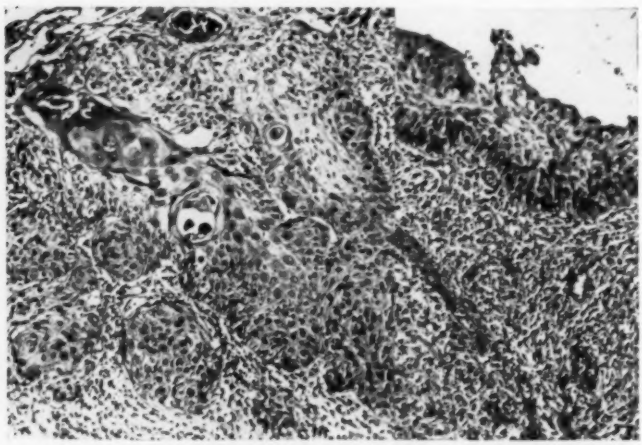
Fig. 4. Pseudosarcoma of larynx. High-power view of the bizarre tumor cells. (H&E x 760.)

Pathology.

Grossly, the tumor consisted of several grayish-pink polypoid structures with glassy, edematous appearance and ulcerated surface. The largest polypoid mass measured approximately 1.5 x 1 cm. in diameter. Microscopically, the bulk of the tumor, on the left side, consisted of interlacing bundles and fascicles of atypical large fibroblasts. In the central portion of the polypoid masses, the interlacing bundles of abnormal fibroblasts showed compact arrangement and formed collagen bundles (see Fig. 1). Toward the periphery and in the immediate vicinity of the ulcerated surface, the tissue was markedly edematous and inflammatory cells abundant (see Fig. 2). Under higher magnification, the fibroblasts showed bizarre giant forms with prominent nuclear anaplasia and numerous mitotic figures (see Figs. 3 and 4). The mitotic figures were frequently atypical. Numerous sections were made to find areas of squamous-cell carcinoma on the surface. The squamous-cell carcinoma was of a well differentiated variety but showed definite infiltration into the underlying pseudosarcomatous stroma (see Fig. 5a). The demarcation between the

two components was usually sharp, but occasionally spindle shaped cells of the pseudosarcomatous stroma were intimately admixed with cells of a squamous-cell carcinoma. Occasionally the surface contained foci of squamous-cell carcinoma *in situ* (see Fig. 5b). The tumor was very vascular, particularly in the periphery; there it showed some resemblance to granulation tissue.

The mucosa from the biopsy on the right vocal cord contained a small focus of carcinoma *in situ*. There was only a small amount of underlying stroma which showed inflammatory cells.



a. b.

Fig. 5. Pseudosarcoma of larynx.
a. Left—Area of invasive well-differentiated squamous-cell carcinoma.
b. Right—Focus of *in-situ* carcinoma in another area. (H&E x 140.)

COMMENT.

Stout[3] in 1957 has also recognized this entity and described similar pedunculated pseudosarcomas in the esophagus. The overlying squamous-cell carcinoma might be, as in our case, invasive, or interepithelial, and usually well differentiated. The reason for this peculiar pseudosarcomatous proliferation of the underlying stroma is unknown. Lane believed the normally loose connective tissue in the oral cavity and larynx might be particularly susceptible to swelling and inflammatory changes from a variety of conditions. The inflammatory

component and increased vascularity of the lesion was also present in our case, and the overlying squamous-cell carcinoma formed only a small portion of the polypoid tumor.

In our patient, as in other patients noted in the literature, with pseudosarcoma associated with carcinoma of the larynx, the important points seem to be:

1. The important lesion is squamous-cell carcinoma which may be inconspicuous, and readily overlooked since the pseudosarcomatous stroma may be abundant and predominant.

2. Although the pseudosarcomatous stroma may appear deceptively like a sarcoma, meticulous study will disclose its benign nature.

3. The lesion should be treated as squamous-cell carcinoma with metastasizing ability.

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Harper Hospital.

LETTER TO THE PATIENT WITH A SENSORI-NEURAL HEARING LOSS.

H. A. Ted Bailey, Jr., M.D.,
and
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We have observed that the largest number of patients seen in our Ear Clinic have been interested in the surgical correction of their hearing loss. Naturally many of these patients have a sensori-neural impairment and are most anxious for surgical help since their problem creates the greater hearing handicap.

In many instances from the moment the sensori-neural patient is told that his type of hearing loss has no surgical alleviation, his acute disappointment greatly interferes with his reception of all further advice and suggestions as to the nature and management of his problem. In addition to this disappointment with its distracting influence, these cases and still others who are not so acutely disappointed may well misunderstand all further advice and instruction because of the very nature of their hearing loss.

Another factor of which we have been very conscious is the possibility that the lack of direct medical treatment may reflect itself to the patient as disinterest on the part of the otologist in his over-all problem.

The above stated reasons have prompted us to prepare a letter to be mailed to the patient with a sensori-neural loss, a few days after he is seen in our Clinic. This affords the opportunity of iteration of our original diagnosis and recommendations at a time when he is more psychologically receptive. It is then possible for the patient to refer to the letter in the future and it will also serve as a source of explanation of his problem not only to himself but also to his family and

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close friends. While we send these letters to all cases of sensori-neural loss, we feel that this is especially contributory to those who are not to return to our Audiology Department for continued guidance.

We have developed two letters which can be mailed to most sensori-neural cases. One is for the patient who can satisfactorily use a hearing aid and the other for the patient who, in our judgment, would not receive sufficient benefit from a hearing aid at the time.

In order for these letters to convey particular interest, in addition to information to the patient, we feel each one must appear as a personal letter. We have accomplished this by having a photograph made of each of the letters. Off-set printing on our stationery then makes it possible to have each letter appear as a personal one when the patient's name, address and salutation are appropriately inserted. The letter is always signed. The above described method of handling these letters satisfies the individual and practical desires of both patient and doctor.

The deep appreciation with which these letters have been received has motivated this presentation.

Letter to the Patient Who Should Wear a Hearing Aid.

"After very careful medical examination and hearing testing, we have determined that your loss of hearing is due to some damage to the hearing nerve itself. Unfortunately, no medical treatment or surgical procedures have been devised to correct this type of problem; nevertheless there is a great deal you can do to cope with your hearing loss.

"When hearing loss is due to damage to the hearing nerve, frequently it is possible for the person to hear sounds but not be able to understand some of the finer sounds of speech. This is certainly confusing and exasperating to him, for he finds it is of little or no help when his friends or relatives shout to him in an attempt to make him understand what they are saying.

"It is the function of the hearing nerve to sort out the

different sounds that are picked up by the ears so that the brain can properly receive them. If the sounds cannot be properly sorted, then speech frequently becomes confused and seems fuzzy and run together.

"When damage occurs to the nerve of hearing it then becomes necessary for that individual to make a conscious effort to do something that people with normal hearing take for granted. That is, to work very hard at using their residual hearing to its fullest advantage. This can best be accomplished by added concentration and by applying extra effort to the art of listening.

"It will be very helpful to you if you watch very carefully the faces of those who speak to you. We are not suggesting lip reading only but the careful watching of all facial expressions. This can frequently make the difference between understanding and not understanding. All people "hear" better when they watch the speaker. This is even more true in your case. Many people who try to "read lips" make the mistake of concentrating on individual words or syllables. Since words are used merely to convey thoughts or ideas, it is far better to concentrate on the general subject of the speaker rather than on isolated sounds or lip movements.

"As in most cases of nerve type hearing impairment, your greatest loss is in the higher tones where the whispered sounds of speech like "p," "sh," "f," and "th" are found. The sounds which have more force occur in the lower range where you hear much better. If you can take the sounds which you can hear but not see on the lips (like the vowel sounds) and add them to those that you can see on the lips but not hear well (like the consonant sounds mentioned), you should be able to join together these sounds so that you can understand much better.

"In many cases of hearing loss due to nerve damage, amplification of sound by means of a hearing aid has proven beneficial. Upon evaluating the results of your hearing examination, especially the speech tests, we feel that it is very probable that you can derive benefit from a hearing aid. The

modern hearing aid, although a wonderful device, is limited in what it can do for a patient with your type of hearing problem. A hearing aid essentially is an amplifier much like that in your television set or radio at home. It can, therefore, be made louder or softer at your discretion, but its microphone will naturally pick up many sounds in your environment in which you are not interested. This frequently may cause you some unhappiness as it will be the speech or music for which you are listening that you wish most to hear, not the slam of a door, rustle of clothes and other such noises. It is only through practice that you can learn to sort out those sounds to which you wish to listen and learn to ignore those unwanted sounds which a hearing aid also amplifies.

"We have tried to paint what we believe to be a realistic picture of your hearing handicap. By making a concentrated effort, it is very likely that you can, in a short time, become a satisfactory user of a hearing aid so that what you can then hear and see will restore you to an easier way of communication with your friends and family and further secure your place in society.

"It is our hope that this explanation has helped to clear up most of the confusing aspects of your hearing loss and that you will feel free to call upon us at any time."

Sincerely,

Letter to the Patient Who Should Not Wear a Hearing Aid.

"After very careful medical examination and hearing testing, we have determined that your loss of hearing is due to some damage to the hearing nerve itself. Unfortunately, no medical treatment or surgical procedures have been devised to correct this type of problem; nevertheless, there is a great deal you can do to cope with your hearing loss.

"When hearing loss is due to damage to the hearing nerve, frequently it is possible for the person to hear sounds but not be able to understand some of the finer sounds of speech. This is certainly confusing and exasperating to him, for he finds it is of little or no help when his friends or relatives

shout to him in an attempt to make him understand what they are saying.

"It is the function of the hearing nerve to sort out the different sounds that are picked up by the ears so that the brain can properly receive them. If the sounds cannot be properly sorted, then speech frequently becomes confused and seems fuzzy and run together.

"When damage occurs to the nerve of hearing it then becomes necessary for that individual to make a conscious effort to do something that people with normal hearing take for granted. That is, to work very hard at using their residual hearing to its fullest advantage. This can best be accomplished by added concentration and by applying extra effort to the art of listening.

"It will be very helpful to you if you watch carefully the faces of those who speak to you. We are not suggesting lip reading only but the careful watching of all facial expressions. This can frequently make the difference between understanding and not understanding. All people "hear" better when they watch the speaker. This is even more true in your case. Many people who try to "read lips" make the mistake of concentrating on individual words or syllables. Since words are used merely to convey thoughts or ideas, it is far better to concentrate on the general subject of the speaker rather than on isolated sounds or lip movements.

"As in most cases of nerve type hearing impairment, your greatest loss is in the higher tones where the whispered sounds of speech like "p," "sh," "f," and "th" are found. The sounds which have more force occur in the lower range where you hear much better. If you can take the sounds which you can hear but not see on the lips (like the vowel sounds) and add them to those that you can see on the lips but not hear well (like the consonant sounds mentioned), you should be able to join together these sounds so that you can understand much better.

"In many cases of hearing loss due to nerve damage, amplification of sound by means of a hearing aid has proven

beneficial. In your case, however, upon evaluating the results of your hearing examination, especially the speech tests, it is our opinion that the benefit you would derive from a hearing aid is not sufficient to justify its use at this time. The modern hearing aid, although a wonderful device, is limited in what it can do for a patient with your type of hearing problem. A hearing aid essentially is an amplifier much like that in your television set or radio at home. It can, therefore, be made louder or softer at the individual's discretion; but in your case its microphone would pick up many sounds in your environment which you do not wish to hear and which would probably make it more difficult for you to understand what you do hear.

"It is our hope that this explanation has helped to clear up most of the confusing aspects of your hearing loss and that you will feel free to call upon us at any time."

Sincerely,

1610 West Third Street.

COURSE IN LARYNGOLOGY AND BRONCHOESOPHAGOLOGY.

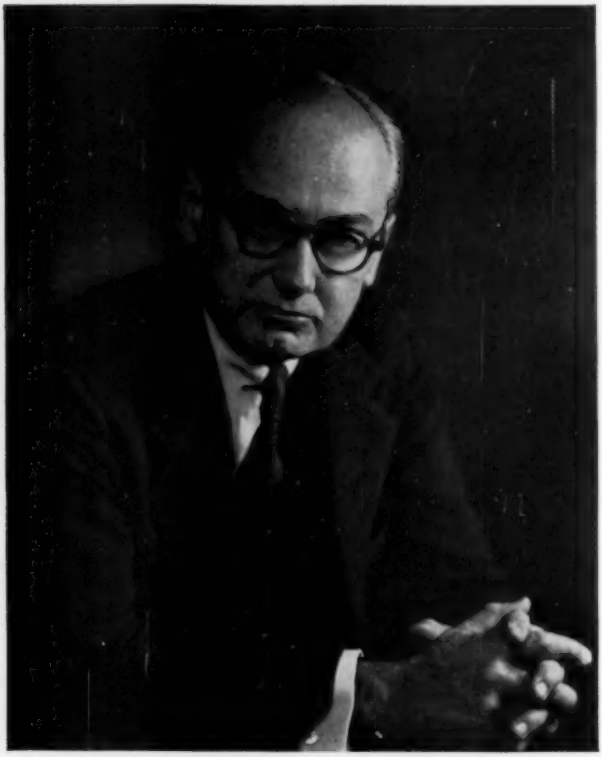
October 23 to November 4, 1961.

The Department of Otolaryngology, University of Illinois College of Medicine, will conduct a postgraduate course in Laryngology and Bronchoesophagology from October 23 through November 4, 1961, under the direction of Paul H. Holinger, M.D.

Registration will be limited to 15 physicians who will receive instruction by means of animal demonstrations and practice in bronchoscopy and esophagoscopy, diagnostic and surgical clinics, as well as didactic lectures.

Interested registrants will please write directly to the Department of Otolaryngology, University of Illinois College of Medicine, 1853 West Polk Street, Chicago 12, Illinois.

In Memoriam



CHEVALIER LAWRENCE JACKSON,
M.D., M.Sc., F.A.C.S., F.C.C.P.

1900-1961

Professor and Head of Department of
Laryngology and Bronchoesophagology,
Temple University Medical Center,
Philadelphia, Pennsylvania.

Chevalier L. Jackson was born in Pittsburgh, August 19, 1900, where he had his early schooling, and he received his Medical Doctorate from the University of Pennsylvania in 1926. His interest in Bronchoscopy developed with his post-graduate studies; however, the supervision of Chevalier Jackson and the teams of associates he had trained intensified his determination to prove himself in his own right, and this zeal and drive continued through his very busy life. Never satisfied to accept the position of the son of a famous father, his internationally recognized career in medicine furthered the research and clinical achievements in laryngology and bronchoesophagology developed by Chevalier Jackson.

"C. L." as he was known to most, "Chevy" to some, and "Lawrence" to a few, received the M.Sc. (Med.) degree from the University of Pennsylvania in 1930 and shortly thereafter accepted an appointment in his specialty at Temple University. He became Professor and Head of the Department of Laryngology and Bronchoesophagology in its School of Medicine in 1946; yet his activities in teaching and organizational medicine were of such international scope that few, if any, can match the breadth of recognition he received and the friendships he made throughout the world. He was invited to give lectures and courses on every continent, and was made an honorary or corresponding member of most national societies of Otolaryngology, Bronchoesophagology, and Diseases of the Chest, of North and South America, Europe and Japan. He was a Commander of the Order of the Liberator San Martin (Argentina), a Chevalier, Legion of Honor (France) and an Honorary Member of the Faculty of Medicine of Universities of Uruguay, Chile and Mexico. In the United States he was a distinguished member of all the national and many regional societies of Otolaryngology and Bronchoesophagology, including the Laennec Society of Philadelphia (President 1941-42), the Philadelphia Laryngological Society (President 1956-58), the American Laryngological Association, the American Laryngological, Rhinological and Otological Society (Vice-President 1957-58), the American Bronchoesophagological Association (President 1950-51), the American Society for Head and Neck Surgery, both the

New York and Philadelphia Bronchoscopic Clubs and the New York Society of Laryngology and Otology. As a Fellow of the American College of Surgeons, Dr. Jackson energetically represented the viewpoint of the laryngologist on its Committee on Cancer of the Larynx as well as on the International Committee for the Study of Cancer of the Larynx, and the Committee on Clinical Stage Classification of the International Union Against Cancer. In Chest circles, Dr. Jackson was as active as he was in Laryngological organizations. He was an early member of the American Association for Thoracic Surgery and a Fellow of the American College of Chest Physicians, serving as its President in 1950-51 and at various times as Chairman of its Bronchoesophagological, Membership, Pan American and International Committees.

In addition to his many scientific contributions, his Committee activities in these organizations were directed toward international participation, and in his extensive travels he invariably carried with him his brief case containing a multiplicity of organizational projects. He founded and served as Secretary of the Pan American Society of Otolaryngology and Bronchoesophagology and the International Broncho-Esophagological Society; was active as Director of the Pan American Medical Association and was a Founder Member of the World Medical Association. His ability to converse in a number of languages was remarkable, and prior to foreign travel he invariably studied the language of the country he intended to visit. His world travels made it very apparent that an International Directory of Otolaryngologists would be helpful and, beginning with a Pan American Directory he later edited an International Directory of Otolaryngology that lists the physicians, national societies, Journals and University faculties of the specialty in all the countries in which this data could be obtained.

Dr. Jackson contributed much to the scientific literature of otolaryngology, including seven textbooks, chapters on peroral endoscopy in other texts, and several hundred articles in the scientific journals of many countries. His interest in segmental bronchial nomenclature, bronchial adenoma, endo-

scopic photography, bronchography, the laryngeal neoplasms, foreign bodies in the air and food passages, and in endoscopic techniques and instrumentarium were presented from a rich clinical experience and a thorough statistical and laboratory study; yet, as is so aptly stated by his former associate and colleague, Mr. Maxwell Ellis, in the British Medical Journal of February 4, 1961, "He held his opinions strongly, and discussions often became argument, sometimes vociferous and turbulent." He adds, "However, one would eventually adjourn to the kitchen to raid the refrigerator and part amicably . . ."

In 1931 Dr. Jackson married Hilda Cowling who survives him with their daughter Joan, her husband Frank Bugbee and their two children Frank and Susan.

"C. L.'s" love of music and opera particularly, was expressed with the same intensity that characterized his medical life. In his own community he took pride in his position of President of the Philadelphia Grand Opera Company. For this and for his internationally recognized medical skills he was held in high esteem and honor by the citizens of Philadelphia as well as by his medical colleagues throughout the world.

His loss will be felt nationally and internationally in civic, medical and photographic circles in all of which his enthusiasm, vision and new ideas were always stimulating.

P. H. H.

BOOK REVIEW.

Through the Barriers of Deafness and Isolation. By Boris V. Morkovin. 178 pages. Macmillan Co., N. Y., 1960.

This volume is the outgrowth of years of active participation in collaboration with Mrs. Lucelia M. Moore in classes and clinic for the hard-of-hearing and deaf at University of Southern California, Los Angeles. Dr. Morkovin is well known for his development of films for use in teaching the deaf and hard-of-hearing. Several teachers of the deaf have collaborated by contributing chapters stressing the teaching of speech skills through a multi-sensory approach and early initiation into the production of normal speech sounds, using films for lip-reading, utilizing many every-day situations for material.

Part I presents a functional approach to speech. Part II deals with the medical, audiological and psychological considerations. Medical findings regarding diseases which cause deafness are discussed, the value of the fenestration operation is described, and the early use of hearing aids recommended.

Part III lays the foundation for oral communication in the home; associating simple patterns made by the speaker's lips with objects; gestures and facial expression. Language experience in the kindergarten in preparation for entering regular classes for the deaf is discussed. Part IV discusses oral communication on the concrete conceptual level, presenting various types of stimulating material for the primary grades. It also includes the use of group hearing aids in classes for children aged eight and nine years. Speech rhythm through music, use of choric speech, singing and dancing are used to make the child aware of and able to use rhythm in speech.

Part V deals with transition to abstract language. Visual aids, pictures, films, school projects and exhibits are recommended. Part VI deals with "integration" of the hearing-impaired child into regular classes with hearing children. Part VII presents speech training by the use of films using life situations, acted out on the screen, to be read from the lips of the actors. Kinesthetic and tactile impressions are also used. Part VIII contains a case history of a boy, deaf from scarlet fever, who became an outstanding professional man despite his deafness, serving as a dental surgeon in a dental college in California.

In Part IX the purposes of the book are summarized, including speech-reading, development of effective oral communication, knowledge of contributions of medicine, electronics and improved hearing aids. An excellent appendix at the end names centers for special hearing services, public residential schools for the deaf, public day classes and American Agencies of the American Hearing Society. The book presents a new orientation in the training of the deaf and hard-of-hearing.

S. S. H.

SEVENTH INTERNATIONAL CONGRESS OF NEUROLOGY.

(Rome, 10-15 September 1961)

FIFTH INTERNATIONAL CONGRESS OF ELECTROENCEPHALOGRAPHY AND CLINICAL NEUROPHYSIOLOGY.

(Rome, 7-13 September 1961).

The Seventh International Congress of Neurology will be held in Rome, September 10-15, 1961, under the auspices of the World Neurologic Federation and of the National Institute for Nervous Diseases and Blindness of Bethesda.

Together with the general Assembly of the International League against Epilepsy and the Vth International Congress of Electroencephalography the first day will be dedicated to the opening Session.

September 11th and 12th will be given respectively to the discussion of Topics I and II, while Topic III and the communications will be represented respectively on the 14th and 15th. Symposia will be held on September 13, a day free of other Congress engagements, to permit an interval between the two parts of the works.

Registration fees and other modalities are the same as those established for the Congress of Neurology. The official banquet and dance will be held in conjunction with this Congress.

Any information is obtainable from the President of the Congress, Prof. Mario Gozzano, or from the Secretary General, Dr. Raffaello Vizioli, Viale Universita 30, Roma.

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES.

(Secretaries of the various societies are requested to keep this information up to date).

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Erling W. Hansen, 90 So. Ninth St., Minneapolis, Minn.
Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.
Meeting: Palmer House, Chicago, Ill., October 8-13, 1961.

AMERICAN ASSOCIATION FOR CLEFT PALATE REHABILITATION.

President: Dr. J. J. Longacre, 1503 Carew Tower, Cincinnati, O.
President-Elect: Dr. D. C. Samuel Pruzansky, D.D.S., 840 So. Wood St., Chicago, Ill.
Secretary-Treasurer: Dr. Spriestersbach, Ph.D., Department of Otolaryngology, University Hospital, Iowa City, Ia.
Meeting:

AMERICAN BOARD OF OTOLARYNGOLOGY.

President: Dr. Gordon D. Hoople, 1100 E. Genesee Dr., Syracuse 10, N. Y.
Secretary: Dr. Dean M. Lierle, University Hospital, Iowa City, Ia.
Meeting: Palmer House, Chicago, Ill., October 2-5, 1961.

AMERICAN BRONCHO-ESOPHAGOLOGICAL ASSOCIATION.

President: Dr. F. Johnson Putney, 1712 Locust St., Philadelphia 3, Pa.
Vice-President: Dr. Julius W. McCall, 619 Rose Bldg., 2060 E. 9th St., Cleveland 15, O.
Secretary: Dr. Daniel C. Baker, Jr., 903 Park Ave., New York 21, N. Y.
Treasurer: Dr. Charles M. Norris, 3401 No. Broad St., Philadelphia 40, Pa.
Meeting: Lake Placid Club, Lake Placid, N. Y., May 23-24, 1961 (afternoons only).

AMERICAN LARYNGOLOGICAL ASSOCIATION.

President: Dr. Edwin N. Broyles, Baltimore, Md.
Secretary: Dr. Lyman G. Richards, Wellesley Hills, Mass.
Treasurer: Dr. Francis E. LeJeune, New Orleans, La.
Editor, Historian, and Librarian: Dr. Francis W. Davison, Danville, Pa.
Meeting: Lake Placid Club, Essex Co., N. Y., May 21-22, 1961.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

President: Dr. Fletcher D. Woodward, 400 Locust Ave., Charlottesville, Va.
President-Elect: Dr. John R. Lindsay, Chicago, Ill.
Secretary: Dr. C. Stewart Nash, 700 Medical Arts Bldg., Rochester 7, N. Y.
Treasurer: Dr. K. M. Day, 121 University Pl., Pittsburgh, Pa.
Annual Meeting: Lake Placid Club, Essex Co., N. Y., May 23-24-25, 1961.

AMERICAN MEDICAL ASSOCIATION, SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY.

Chairman: Dr. Lawrence R. Boies, Minneapolis, Minn.
Vice-Chairman: Dr. Howard P. House, Los Angeles, Calif.
Secretary: Dr. Walter E. Heck, San Francisco, Calif.
Delegate: Dr. Gordon F. Harkness, Davenport, Ia.
Alternate Delegate: Dr. Paul H. Holinger, Chicago, Ill.
Representative to Scientific Exhibit: Dr. Walter H. Maloney, Cleveland, O.
Meeting: New York City, June 26-30, 1961.

AMERICAN OTOLOGICAL SOCIETY, INC.

President: Dr. Henry L. Williams, Rochester, Minn.
Vice-President: Dr. Lawrence R. Boies.
Secretary-Treasurer: Dr. James A. Moore, New York City, N. Y.
Annual Meeting: Lake Placid Club, Essex Co., N. Y., May 26-27, 1961.

AMERICAN OTORHINOLOGIC SOCIETY FOR THE ADVANCEMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY.

President: Dr. Joseph Gilbert, 111 E. 61st St., New York, N. Y.
Vice-President: Dr. Kenneth Hinderer, 402 Medical Arts Bldg., Pittsburgh, Pa.
Secretary: Dr. Louis Joel Feit, 66 Park Ave., New York 16, N. Y.
Treasurer: Dr. Arnold L. Caron, 36 Pleasant St., Worcester, Mass.

AMERICAN RHINOLOGIC SOCIETY.

President: Dr. Ivan W. Philpott, 1801 High St., Denver, Colo.
Secretary: Dr. Robert M. Hansen, 2210 Lloyd Center, Portland 12, Ore.
Annual Clinical Session: Chicago, Ill., October, 1961 (definite time and place to be announced later).
Annual Meeting: Chicago, Ill., October, 1961 (definite time and place to be announced later).

AMERICAN SOCIETY FOR HEAD AND NECK SURGERY.

President: Dr. John J. Conley, New York, N. Y.
Vice-President: Dr. Joseph H. Ogura, St. Louis, Mo.
Treasurer: Dr. F. Johnson Putney, Philadelphia, Pa.
Secretary: Dr. George A. Sisson, Syracuse, N. Y.
Annual Meeting: Palmer House, Chicago, Ill.

AMERICAN SOCIETY OF FACIAL PLASTIC SURGERY.

President: Dr. Sam H. Sanders, 1089 Madison Ave., Memphis, Tenn.
Vice-President: Dr. John T. Dickinson, Mercy Hospital, Pittsburgh 19, Pa.
Secretary: Dr. Samuel M. Bloom, 123 East 83rd St., New York 28, N. Y.
Treasurer: Dr. Joseph C. Miceli, 879 Glenmore Ave., Brooklyn, N. Y.
Winter Meeting: February 9-10, 1961, Peabody Hotel, Memphis, Tenn.
Summer Meeting: June 28, 1961, Hotel Elysee, New York, N. Y.

AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY.

President: Dr. Leland H. Prewitt, Ottumwa, Ia.
Secretary-Treasurer: Dr. Daniel S. DeStio, 121 S. Highland Ave., Pittsburgh 6, Pa.
Annual Meeting: Palmer House, Chicago, Ill., October 7, 1961.

ASSOCIACAO MEDICA DO INSTITUTO PENIDO BURNIER—CAMPINAS.

President: Dr. Alberto Gallo.
First Secretary: Dr. Alfredo Martinelli.
Second Secretary: Dr. Guedes de Melo Neto.
Librarian-Treasurer: Dr. L. de Souza Queiroz.
Editors for the Archives of the Society: Dr. Antonio de Almeida, Dr. Gabriel Pôrto, and Dr. Roberto Franco do Amaral.

ASOCIACION DE OTORRINOLARINGOLOGIA Y BRONCOESOFAGOLOGIA DE GUATEMALA.

Presidente: Dr. Julio Quevedo, 15 Calle Oriente No. 5.
First Vice-Presidente: Dr. Héctor Cruz, 3a Avenida Sur No. 72.
Second Vice-Presidente: Dr. José Luis Escamilla, 5a Calle Poniente No. 48.
Secretario-Tesorero: Dr. Horace Polanco, 13 Calle Poniente No. 9-D.

ASOCIACION DE OTO-RINO-LARINGOLOGIA DE BARCELONA, SPAIN.

Presidente: Dr. J. Abello.
Vice-Presidente: Dr. Luis Sufie Medan.
Secretario: Dr. Jorge Perellò, 319 Provenza, Barcelona.
Vice-Secretario: Dr. A. Pinart.
Vocal: Dr. J. M. Ferrando.

BALTIMORE NOSE AND THROAT SOCIETY.

Chairman: Dr. Walter E. Loch, 1039 No. Calvert St., Baltimore, Md.
Secretary-Treasurer: Dr. Theodore A. Schwartz.

BUENOS AIRES CLUB OTOLARINGOLOGICO.

Presidente: Dr. K. Segre.
Vice-Presidente: Dr. A. P. Belou.
Secretario: Dr. S. A. Aranz.
Pro-Secretario: Dr. J. M. Tato.
Tesorero: Dr. F. Games.
Pro-Tesorero: Dr. J. A. Bello.

CANADIAN OTOLARYNGOLOGICAL SOCIETY SOCIETE CANADIENNE D'OTOLARYNGOLOGIE.

President: Dr. Fernand Montreuil, 1123 St. Joseph Blvd. East, Montreal, Quebec.
Secretary: Dr. Donald M. MacRae, 324 Spring Garden Road, Halifax, Nova Scotia.
Meeting: Queen Elizabeth Hotel, June 15-17, 1961.

CENTRAL ILLINOIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. G. E. Hartenbower, 203 N. Main St., Bloomington, Ill.
President-Elect: Dr. Edgar T. Blair, Springfield, Ill.
Vice-President: Dr. G. LeRoy Porter, Urbana, Ill.
Delegate at Large: Dr. S. G. Baldwin, Danville, Ill.
Secretary-Treasurer: Dr. C. L. Pannabecker, Peoria, Ill.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

President: Dr. George Woodruff, Woodruff Clinic, Joliet, Ill.
Vice-President: Dr. Linden Wallner, 122 So. Michigan, Chicago, Ill.
Secretary-Treasurer: Dr. Robert Lewy, 25 East Washington St., Chicago 2, Ill.
Meeting: First Monday of each month, October through May.

CHILEAN SOCIETY OF OTOLARYNGOLOGY.

President: Dr. Enrique Grünwald S.
Vice-President: Dr. Agustin Estartus.
Secretary: Dr. Marcos Chaimovich S.
Treasurer: Dr. Benjamin Kapkan K.
Director: Dr. Alberto Basterrica A.

COLORADO OTOLARYNGOLOGY SOCIETY.

President: Dr. James T. Blair, Denver, Colo.
Vice-President: Dr. James Rigg, Grand Junction, Colo.
Secretary: Dr. Will P. Pirkey, Denver, Colo.

COLUMBUS, OHIO, OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY.

President: Dr. John E. Arthur.
Secretary: Dr. M. L. Battles.
Meetings: First Monday of October through May, University Club, Columbus, O.

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Edward A. Newell.
Vice-President: Dr. Thomas M. McCrory.
Secretary-Treasurer: Dr. James L. Baldwin, 1627 Medical Arts Bldg., Dallas, Tex.

FEDERACION ARGENTINA, DE SOCIEDADES DE OTORRINOLARINGOLOGIA.

Secretary of the Interior: Prof. Dr. Atilio Viale del Carril.
Secretary of the Exterior: Dr. Aldo G. Remorino.
Secretary Treasury: Prof. Dr. Antonio Carrascosa.
Pro-Secretary of the Interior: Prof. Dr. Carlos P. Mercandino.
Pro-Secretary of the Exterior: Prof. Dr. James A. del Sel.
Pro-Secretary of the Treasury: Dr. Jorge Zubizarreta.

FIRST CENTRAL AMERICAN CONGRESS OF OTORHINOLARYNGOLOGY.

President: Dr. Victor M. Noubleau, San Salvador.
Secretary-Treasurer: Dr. Hector R. Silva, Calle Arce No. 84, San Salvador, El Salvador, Central America.

FLORIDA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. G. Dekle Taylor, Jacksonville, Fla.
President-Elect: Dr. Kenneth S. Whitmer, Miami, Fla.
First Vice-President: Dr. William H. Anderson, Jr., Ocala, Fla.
Second Vice-President: Dr. Marion W. Hester, Lakeland, Fla.
Secretary-Treasurer: Dr. Joseph W. Taylor, Jr., 1 Davis Blvd., Tampa 6, Fla.

FOURTH LATIN-AMERICAN CONGRESS OF OTORINOLARINGOLOGIA.

President: Dr. Dario.
Secretary:
Meeting:

FORT WORTH EYE, EAR, NOSE AND THROAT SOCIETY.

President: Dr. Van D. Rathgeber.
Vice-President: Dr. William Skokan.
Secretary-Treasurer: Dr. Paul Rockwell.

GEORGIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. P. W. Rhyne, Albany, Ga.
Vice-President: Dr. C. L. Pennington, Macon, Ga.
Secretary: Dr. James T. King, 516 Baptist Professional Bldg., Atlanta 12, Ga.
Meeting: Oglethorpe Hotel, Wilmington Island, Savannah, Ga., March 2-4, 1961.

GREATER MIAMI EYE, EAR, NOSE AND THROAT SOCIETY.

President: Dr. Mariano C. Caballero.
Vice-President: Dr. Joseph Freeman.
Secretary-Treasurer: Dr. H. Carlton Howard.
Meeting: Quarterly in March, May, October and December on the second Thursday of the month, 6:30 P.M., at the McAllister Hotel, Miami, Fla.

INTERNATIONAL BRONCHOESOPHAGOLOGICAL SOCIETY.

President: Dr. Giorgio Ferreri, Rome, Italy.
Acting Secretary: Dr. Charles M. Norris, 3401 N. Broad St., Philadelphia 40, Pa., U. S. A.
Meeting: Tenth International Congress of Bronchoesophagology, Reims, France and Dusseldorf, Germany, July 30-August 2, 1961.

KANSAS CITY SOCIETY OF OTOLARYNGOLOGY AND OPHTHALMOLOGY.

President: Dr. Clarence H. Steele.
President-Elect: Dr. Dick H. Underwood.
Secretary: Dr. James T. Robison, 4620 J. C. Nichols Parkway, Kansas City, Mo.
Meeting: Third Thursday of November, January, February and April.

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Max E. Pohlman.
Secretary-Treasurer: Dr. Wendell C. Irvine.
Chairman of Ophthalmology Section: Dr. Carroll A. McCoy.
Secretary of Ophthalmology Section: Dr. Philip D. Shanedling.
Chairman of Otolaryngology Section: Dr. Robert W. Godwin.
Secretary of Otolaryngology Section: Dr. Francis O'N. Morris.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd., Los Angeles, Calif.
Time: 6:30 P.M. last Monday of each month from September to June, inclusive—Otolaryngology Section. 6:30, first Thursday of each month from September to June, inclusive—Ophthalmology Section.

LOUISIANA-MISSISSIPPI OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY.

President: Dr. Carl E. Granberry.
Secretary: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.
Meeting: Edgewater Gulf Hotel, Edgewater Park, Miss., April 13-14, 1962.

MEMPHIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Chairman: Members serve as chairman in alphabetical order monthly.
Secretary-Treasurer: Dr. Roland H. Myers, 1720 Exchange Bldg., Memphis, Tenn.
Assistant Secretary-Treasurer: Dr. William F. Murrah, Jr., Exchange Bldg., Memphis, Tenn.
Meeting: Second Tuesday in each month at 8:00 P.M. at Memphis Eye, Nose and Throat Hospital.

MEXICAN ASSOCIATION OF PLASTIC SURGEONS.

President: Dr. Cesar LaBoide, Mexico, D. F.
Vice-President: Dr. M. Gonzales Ulloa, Mexico, D. F.
Secretary: Dr. Juan De Dios Peza, Mexico, D. F.

MEXICAN SOCIETY OF OTOLARYNGOLOGY.

President: Dr. Rafael Giorgana.
Secretary: Dr. Carlos Valenzuela, Petrarca 332-1, Mexico 5, D. F.

THE MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY.

President: Dr. Paul G. Bunker, Aberdeen, S. Dak.
First Vice-President: Dr. John A. McNeill, St. Paul, Minn.
Second Vice-President: Dr. Benjamin Bofenkamp, Minneapolis, Minn.
Secretary-Treasurer: Dr. Richard O. Leavenworth, Jr., St. Louis Park, Minn.

MISSISSIPPI VALLEY MEDICAL SOCIETY.

President: Dr. Arthur S. Bristow, Princeton, Mo.
Secretary-Treasurer: Dr. Harold Swanberg, Quincy, Ill.
Assistant Secretary-Treasurer: Dr. Jacob E. Reisch, Springfield, Ill.

NETHERLANDS SOCIETY OF OTO-RHINO-LARYNGOLOGY.
(Nederlandsche Keel-Neus-Oorheelkundige Vereeniging.)

President: Dr. H. Navis, Sonsbeekweg 6, Arnhem.
Secretary: Dr. W. H. Struben, J. J. Viottastraat 1, Amsterdam.
Treasurer: Mrs. F. Velleman-Pinto, Jac. Ohrechtstr. 66, Amsterdam.

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY.

President: Dr. George T. Noel, Kannapolis, N. C.
Vice-President: Dr. J. C. Bradley, Weaverville, N. C.
Secretary: Dr. John S. Gordon, 1350 South Kings Drive, Charlotte, N. C.

NORTH OF ENGLAND OTOLARYNGOLOGICAL SOCIETY.

President: Mr. G. L. Thompson, 16 Ramshill Road, Scarborough, Yorkshire.
Vice-President: Mr. J. H. Otty, Frizley Old Hall, Frizinghall Road, Bradford, Yorkshire.
Secretary and Treasurer: Mr. R. Thomas, 27 High Petergate, York, Yorkshire.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. George C. Saunders, 644 Medical Arts Bldg., Portland 5, Ore.
Secretary-Treasurer: Dr. Donald C. Mettler, 1216 S. W. Yamhill St., Portland 5, Ore.
Meeting: Fourth Tuesday of each month from September through May, Aero Club, Portland, Ore.

OTOSCLEROSIS STUDY GROUP.

President: Dr. Arthur L. Juers, 1018 Brown Bldg., Louisville, Ky.
Vice-President: Dr. Victor Goodhill, 4759 Hollywood Blvd., Los Angeles 27, Calif.
Secretary-Treasurer: Dr. R. E. Jordan, 3515 Fifth Ave., Pittsburgh 13, Pa.
Meeting: Palmer House, Chicago, Ill., October, 1961.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY.

President: Dr. John F. Tolan, 1118 - 9th Ave., Seattle 5, Wash.
Secretary-Treasurer: Dr. Homer E. Smith, 686 Twelfth Ave., Salt Lake City, Utah.

PAN AMERICAN ASSOCIATION OF OTO-RHINO-LARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY.

President: Dr. Plino de Mattos Barretto, Sao Paulo, Brazil.
Acting Executive Secretary: Dr. Charles M. Norris, 3401 No. Broad St., Philadelphia 40, Pa., U. S. A.
Meeting: Eighth Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology, Puerto Azul Club, Valencia, Venezuela, S. A., February 25th to March 1st, 1962.

PHILADELPHIA LARYNGOLOGICAL SOCIETY.

President: Dr. Joseph P. Atkins.
Vice-President: Dr. Louis E. Silcox.
Secretary: Dr. Charles M. Norris.
Treasurer: Dr. William A. Lell.

PHILIPPINE SOCIETY OF OTOLARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY.

President: Dr. Cesar F. Villafuerte.
Vice-President: Dr. Napoleon C. Ejercito.
Secretary-Treasurer: Dr. Eusebio E. Llamas.
Directors: Dr. Antonio L. Roxas and Dr. Armando T. Chiong.

PITTSBURGH OTOLOGICAL SOCIETY.

President: Dr. Emory A. Rittenhouse, 203 Masonic Bldg., McKeesport, Pa.
Vice-President: Dr. Carson S. Demling, 513 Jenkins Bldg., Pittsburgh 22, Pa.
Secretary-Treasurer: Dr. Clyde B. Lamp, 3101 Jenkins Arcade, Pittsburgh 22, Pa.

PORTUGUESE OTORHINOLARYNGOLOGICAL SOCIETY.

President: Dr. Albert Luis de Mendonca.
Secretary: Dr. Antonio da Costa Quinta, Avenida, de Liberdale 65, 1° Lisbon.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Clifton E. Benson, Bremerton, Wash.
President-Elect: Dr. Carl D. F. Jensen, Seattle, Wash.
Secretary: Dr. Willard F. Goff, 1215 Fourth Ave., Seattle, Wash.

SEVENTH INTERNATIONAL CONGRESS ON DISEASES OF THE CHEST.

RESEARCH STUDY CLUB OF LOS ANGELES, INC.

Chairman: Dr. Orrie E. Ghrist, 210 N. Central Ave., Glendale, Calif.
Treasurer: Dr. Norman Jesberg, 500 So. Lucas Ave., Los Angeles 17, Calif.
Otolaryngology: Dr. Russell M. Decker, 65 N. Madison Ave., Pasadena 1, Calif.
Ophthalmology: Dr. Warren A. Wilson, 1930 Wilshire Blvd., Los Angeles 57, Calif.
Mid-Winter Clinical Convention annually, the last two weeks in January at Los Angeles, Calif.

SECTION ON OTOLARYNGOLOGY OF THE MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA.

Chairman: Dr. Morris E. Krucoff.
Vice-Chairman: Dr. Max J. Fischer.
Secretary: Dr. Adrian J. Delaney.
Treasurer: Dr. Robert D. Ralph.
Meetings are held the second Tuesday of September, November, January, March and May, at 6:30 P.M.
Place: Army and Navy Club, Washington, D. C.

SCOTTISH OTOLARYNGOLOGICAL SOCIETY.

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